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ROENTGENOLOGIC FINDINGS IN THE LUNGS OF VICTIMS OF THE COCOANUT GROVE DISASTER*†

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THE clinical features of the respiratory tract injuries among the victims of the Cocoanut Grove disaster as observed at the Boston City Hospital have been described in detail elsewhere.[1] The severity of the damage to the respiratory tract in these cases was graded roughly according to the clinical manifestations as follows:

Grade	*Clinical Manifestations*
0	No signs or symptoms referable to the respiratory tract.
1	Signs and symptoms limited to the upper respiratory passages, including mild laryngitis.
2	Evidence of involvement of the trachea and bronchi or abnormal signs in the lungs other than those of frank consolidation.
3	Dyspnea, cyanosis, wheezing or transient stridor or frank signs of consolidation.
4	Evidence of extensive pulmonary involvement or of obstruction to the airways necessitating tracheotomy or resulting in severe asphyxia.

The severity of the respiratory damage classified in this way was shown to be correlated fairly closely with the extent of the surface burns. In individual cases, however, there were wide discrepancies but most of these could be explained by unusual circumstances. For example, in some of the patients who died with severe respiratory tract damage but with few or no surface burns, it was ascertained that they were stampeded early and their bodies were protected from the flames by the bodies of others. They nevertheless continued to inhale the hot and irritating fumes. In others who succeeded in extricating themselves from the building or in those who covered their mouth and nose, there was little or no respiratory tract involvement but they sustained burns which were often quite extensive.

The deaths were of two general types and were associated either with (1) burns involving 30 per cent or more of the body surface or (2) severe respiratory tract injuries which resulted in obstruction to the air passages. There were only 2 recoveries among 30 patients with respiratory symptoms of Grade 4 severity.

The signs and symptoms referable to the respiratory tract were interpreted as consistent with the effects of laryngotracheobronchitis of varying extent and severity.

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The autopsy findings suggested further that the clinical manifestations in the severe cases had resulted from varying degrees of obstruction due to the pseudomembrane and the viscid exudate which covered the tracheobronchial tree and often extended down to the small bronchioles. These gave rise to patches of atelectasis in some parts of the lung while in other parts there were areas of emphysema and dilated bronchi due to air trapped beyond the obstructing lesions. In addition, the autopsies revealed thrombosis of pulmonary vessels with infarcted areas in the lungs of patients who died within the first three or four days, and patchy or confluent lesions of bronchopneumonia in some of those who died after that time. As might be expected, these changes gave rise to rather variable and often extensive and bizarre changes in the roentgenographic appearance of the lungs.

Similar but less extensive findings resulting from exposure to noxious gases have been described before.[2,3] A thorough roentgenologic report of the pulmonary lesions in the cases from the Cocoanut Grove fire which were studied at the Massachusetts General Hospital has been given by Schatzki.[4] The clinical features of those cases were described by Aub *et al.*[5] Since Schatzki's descriptions cover most of the changes observed in the cases studied at the Boston City Hospital, they will be summarized here and will serve as a background for the report of roentgen findings observed. The latter will be correlated, in a general way, with the clinical signs and symptoms and with the autopsy findings. Selected roentgenograms in some of the severe cases will serve to illustrate the various types of abnormalities that were seen.

Schatzki found roentgen changes in the lungs of 22 of 35 patients examined. The first roentgenograms showed flame-like areas radiating asymmetrically from both hila with scattered bands and lines, as well as large homogeneous and small miliary areas of increased density. From the further development of these lesions and from the autopsy findings, Schatzki felt that interference with aeration due to complete or partial obstruction of the bronchi, particularly the smaller ones, was the cause of most, if not all of the visible pulmonary changes.

In chest roentgenograms of 2 autopsied cases dead on arrival he found extensive, diffuse, poorly defined haziness extending throughout most of both lung fields. This was interpreted as characteristic of pulmonary edema and that was confirmed by the autopsy findings. Both of these cases showed high concentrations of carbon monoxide in their blood. The roentgenograms in 3 other patients who died in the hospital all showed areas of emphysema and patches of atelectasis. In 1 of them there was evidence of pulmonary edema in the last roentgenogram; another showed a peculiar mottling and the third showed a round area of localized emphysema resembling trapped air such as that which is sometimes seen in children (pneumatocele). Autopsies in these 3 cases showed tracheobronchitis and bronchiolitis with patchy atelectasis and emphysema, some pulmonary edema as well as localized areas of atelectasis and hemorrhage which may have represented early infarctions.

Interestingly enough one of the 13 patients with negative roentgen findings died of respiratory embarrassment some hours after the negative roentgenogram was obtained. Furthermore, all but two of the surviving roentgenologically negative patients had râles on physical examination on one or more occasions.

Schatzki's descriptions of the various types of roentgenographic lesions may be summarized briefly. *Atelectasis* was recognized in several forms. Sudden massive lobar atelectasis was seen in only 1 case, and the gradual development of complete collapse of both lower lobes was observed in another. More commonly there were smaller areas represented by triangles, or by bands, discs, or even fine lines of increased density which ran horizontally, obliquely and, in the lower lobes, even vertically through the lung fields. The oc-

currence, in the same roentgenograms, of compensatory emphysema, elevation of the diaphragm and displacement and contraction of the hilar shadows on the side where the changes were extensive was taken as proof of the atelectactic origin of the shadows. Lateral shift of the mediastinum was rarely seen due to the fact that there was bilateral involvement in most cases.

Emphysema, lobar and lobular in distribution, was seen in several patients during various stages. Schatzki was able to demonstrate them best on roentgenograms taken in expiration, indicating the presence of trapped air. There was usually some atelectasis in the same lungs. One case of persistent lobar emphysema was also described. *Miliary mottling* of both lungs with individual lesions measuring 2 to 6 mm. in diameter was observed in only 2 fatal cases. These shadows were not adequately explained but were thought to be due to small areas of atelectasis or possibly to small plugged bronchi. A homogeneous ground-glass appearance of a large portion of one lobe or one lung without decrease in size of the involved part was noted in the early roentgenograms in 3 survivors. Since these cleared rapidly they were interpreted as representing fluid in partially atelectatic lungs, the so-called "drowned lungs." *Pulmonary edema* was identified in only the 2 fatal cases already mentioned. *Infarcts* of the lungs, though seen in the autopsies, were not recognized roentgenologically except in the later roentgenograms of some of the severely burned cases in which they were obviously connected with thrombophlebitis.

In the cases admitted from the Cocoanut Grove disaster to the Boston City Hospital roentgenograms were not made until about thirty-six hours after the fire except in a few instances. At that time a roentgenogram of the chest was taken at the bedside on almost every patient who was still in the hospital. Additional roentgen examinations of the chest, varying from one to five in number, were made in 57 of the cases during the remainder of the hospital stay. In some of these cases roentgenograms were taken both in inspiration and in expiration. Roentgenograms of the chest were also included as part of the follow-up examination of those who returned to the hospital for this purpose.

After all of the roentgenograms were reviewed, the extent of the abnormal findings in each case was graded according to an arbitrary scale from 0 to 4. Those with the most extensive changes were classified as Grade 4 and those with only minor abnormalities in the roentgenograms were classed as Grade 1. A correlation of the roentgen changes classified in this manner with the severity of the symptoms in each case is shown in Table 1. There was a fairly close correlation between the severity of the respiratory symptoms and the extent of the roentgen changes. Wide discrepancies were very few. In about one-third of the cases the severity of the respiratory symptoms was somewhat greater than was indicated by the roentgen changes in the lungs, but

TABLE I

CORRELATION OF SEVERITY OF RESPIRATORY INVOLVEMENT AS OBSERVED CLINICALLY WITH THE DEGREE OF PULMONARY CHANGES OBSERVED ROENTGENOGRAPHICALLY

Grade of Respiratory Involvement (Clinical)	Degree of Pulmonary Changes Observed in Roentgenograms					
	0	1	2	3	4	Total (cases)
0	8^{1}	2	0	0	0	10^{1}
1	8	14^{1}	6	0	0	28^{1}
2	0	8^{1}	8	1^{1}	0	17^{2}
3	0	2	10	7^{1}	1^{1}	20^{2}
4	0	0	2^{2}	3^{3}	7^{4}	12^{9}
Total (cases)	16^{1}	26^{2}	26^{2}	11^{5}	8^{5}	87^{15}

Six cases are included in which roentgenograms were taken at other hospitals to which the patient was removed within 36 hours after the fire.

44 cases (51%) had same rating clinically and roentgenographically.

29 cases (33%) had a clinical rating 1 grade higher than the roentgen rating.

10 cases (11%) had a roentgen rating 1 grade higher than the clinical rating.

4 cases (5%) had a clinical rating 2 grades higher than the roentgen rating.

Superscripts represent fatal cases.

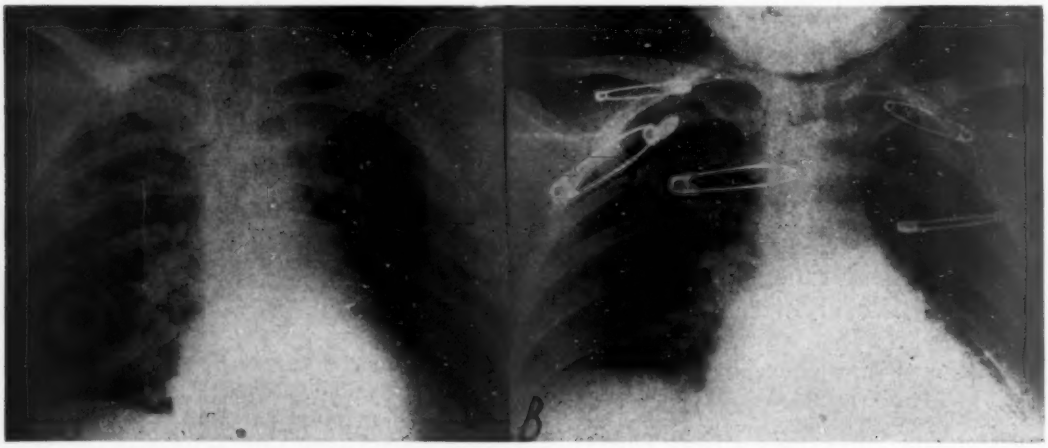

FIG. 1. Case 1. *A*, second day: extensive mottling especially in the right upper lung field; patchy areas of emphysema, and diffusely diminished aeration of the left lower lung. *B*, eleventh day: considerable clearing with diffuse emphysema in the upper lung fields.

The patient lost consciousness early at the fire. On admission she had regained consciousness but was restless and coughing. A few crepitant râles were heard at the base of the right lung and there were cardiac signs of mitral stenosis. Burns involved mostly the face, shoulders and hands—estimated 7 per cent. She rapidly developed hoarseness, dyspnea and her cough markedly increased; she began to raise sputum which was thick, mucoid and black at first and then became blood streaked. When *A* was taken there were showers of loud crepitant râles throughout both lungs and the patient was very dyspneic. The râles decreased gradually during the next few days and only a few were heard over the left lower lobe when *B* was taken. The lungs had cleared completely but there was still some cough and hoarseness when the patient was discharged on the sixteenth day. Three months later she was still having intermittent slight hoarseness and cough but she had improved considerably. One year later her general condition was excellent, she had no more cough but did have a chronic post-nasal discharge which began after the fire. She became hoarse only after using her voice excessively. Roentgen and physical examination of her chest were completely negative.

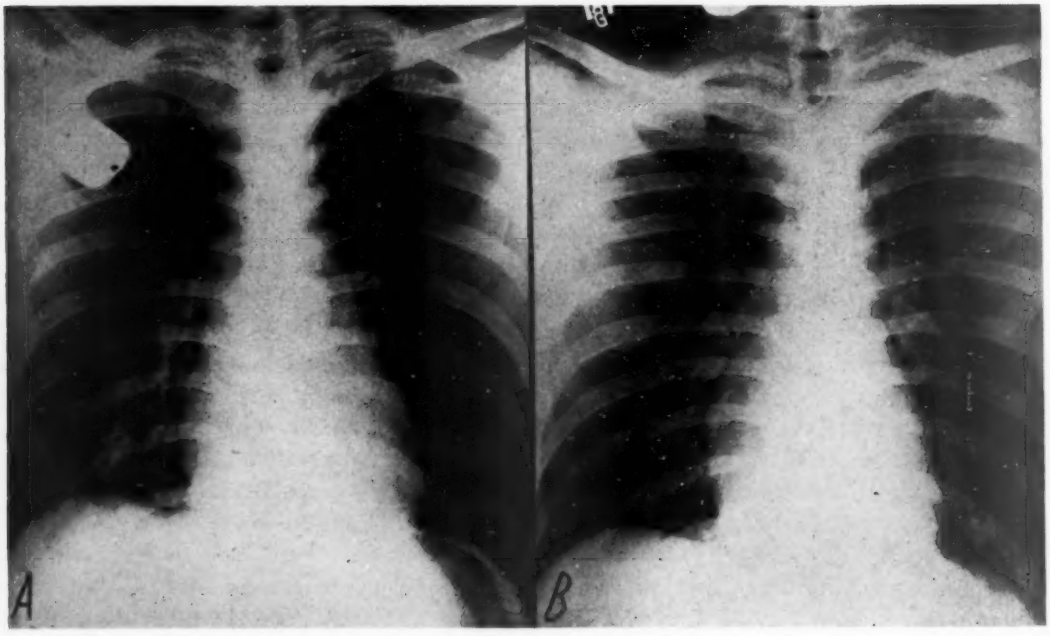

FIG. 2 (*See opposite page for legend.*)

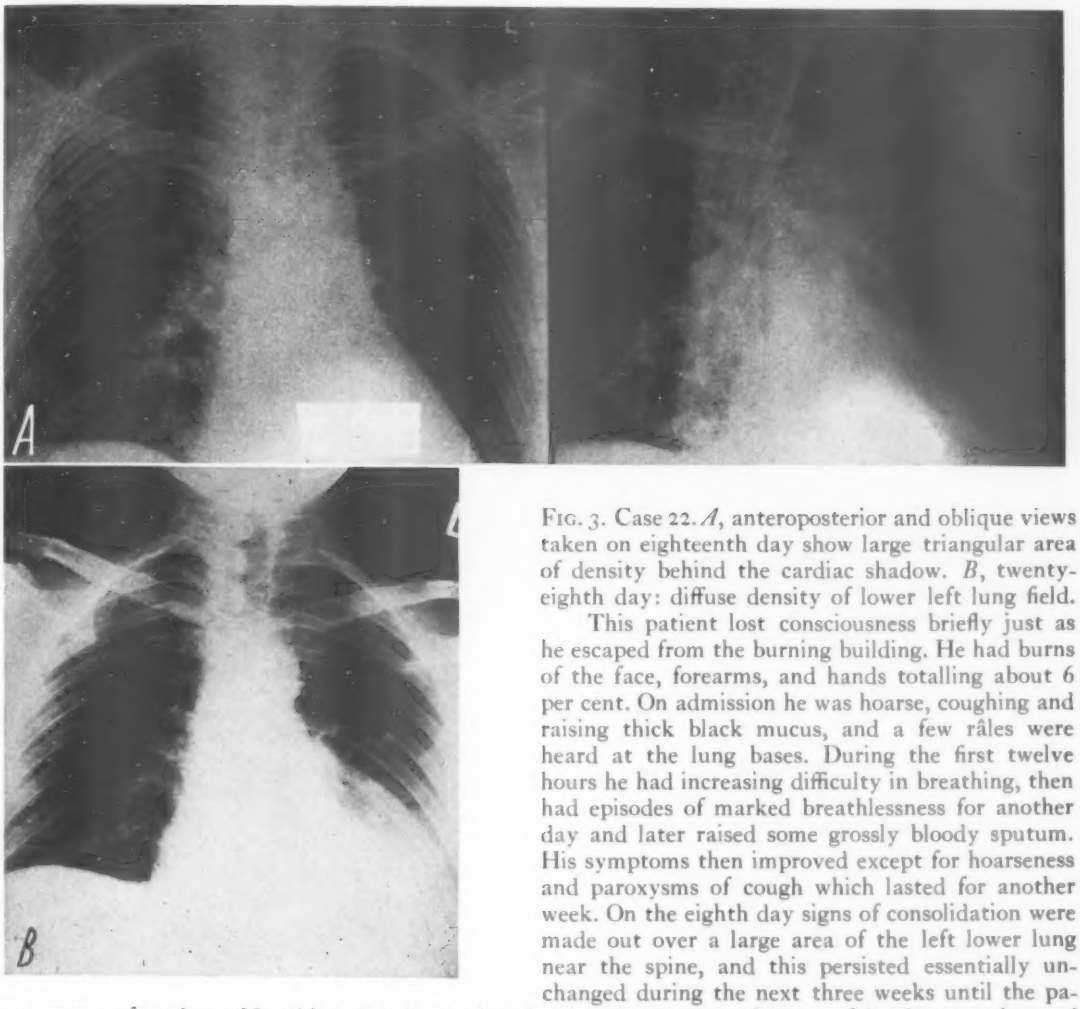

Fig. 3. Case 22. *A*, anteroposterior and oblique views taken on eighteenth day show large triangular area of density behind the cardiac shadow. *B*, twenty-eighth day: diffuse density of lower left lung field.

This patient lost consciousness briefly just as he escaped from the burning building. He had burns of the face, forearms, and hands totalling about 6 per cent. On admission he was hoarse, coughing and raising thick black mucus, and a few râles were heard at the lung bases. During the first twelve hours he had increasing difficulty in breathing, then had episodes of marked breathlessness for another day and later raised some grossly bloody sputum. His symptoms then improved except for hoarseness and paroxysms of cough which lasted for another week. On the eighth day signs of consolidation were made out over a large area of the left lower lung near the spine, and this persisted essentially unchanged during the next three weeks until the patient was transferred to a Naval hospital. In routine chest roentgenograms the area of density was obscured by the cardiac shadow but oblique views showed a dense triangular area extending from the hilum to the diaphragm as seen in the figure. During the fourth week there was an episode of fever associated with extension of the signs throughout the left lower lobe as shown in *B*. The lung has cleared slowly and the patient has been returned to active duty but details of his course after leaving the hospital could not be ascertained.

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Fig. 2. Case 8. *A*, second day: diffuse emphysema; patchy atelectasis in lower right and soft mottling in lower left lung field. *B*, sixth day: residual patchy atelectasis in right lower lung field.

The patient was overcome inside the burning building and regained consciousness while being brought to the hospital. His burns involved the entire face and both ears—total 4 per cent. On arrival he was very hoarse and coughing and numerous râles were heard throughout both lungs. At the time when the earlier roentgenogram was taken he was having dyspnea, occasional episodes of stridor and some pain in the right chest and was raising green sputum containing leukocytes, epithelial cells and type 3 pneumococci. Improvement in symptoms began on the next day and the signs in the lungs cleared progressively so that at the time of the later roentgenogram there were only a few crepitant râles at the bases and occasional scattered high pitched musical râles throughout both lungs. These were still present at the time of discharge three weeks later. He was returned to active duty in the Navy and when last heard from, two years after the fire, he was free of symptoms except for occasional cough productive of a small amount of sputum.

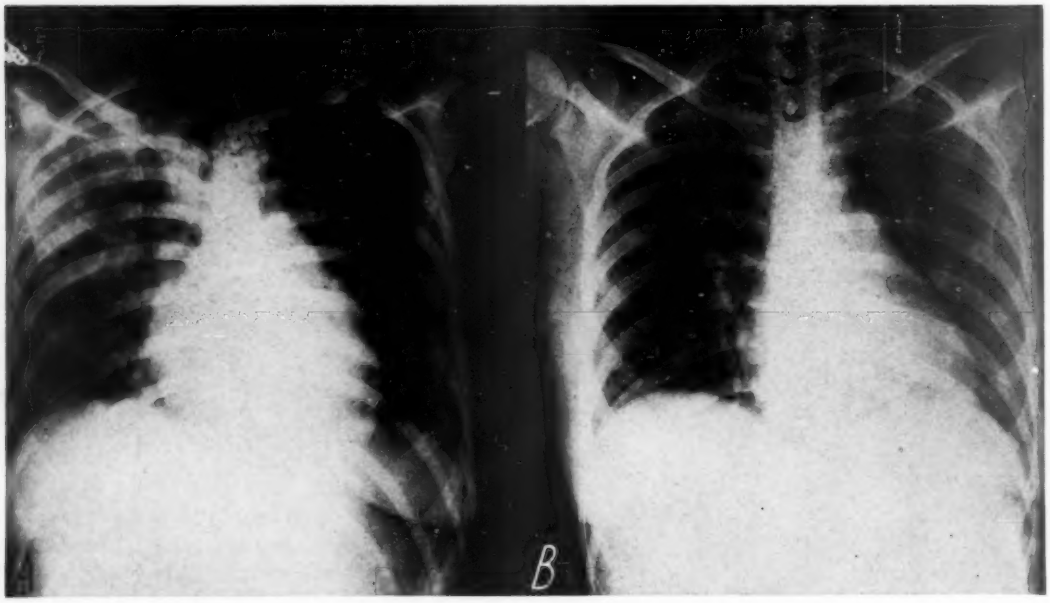

FIG. 4. Case 47. *A*, second day: extensive densities mostly in the right upper lung field with some emphysema (trapped air) in that area. *B*, tenth day: almost complete clearing.

The patient was knocked down and trampled and lost consciousness before she could attempt to escape. She suffered no external burns. On admission she was hoarse and coughing and râles were heard over the right chest. The lungs cleared somewhat for a while but then the patient began to have dyspnea, wheezing, a feeling of constriction in the chest and stridor. A tracheotomy was contemplated but not done when laryngoscopy revealed singed vocal cords. At the time of the earlier roentgenogram the patient was very hoarse and there were showers of crepitant râles at the lung bases and scattered high pitched musical râles, especially in the left lung. The coughing and hoarseness had improved considerably, the crepitant râles had cleared but the high pitched squeaks persisted and the patient was still having difficulty in taking a deep breath at the time when the later roentgenogram was taken. She continued to have some cough and some dyspnea on exertion for the first three months after discharge but after eight months there was only occasional cough and her general condition has been good. Physical examination and roentgenogram of the chest after eight months were entirely negative.

the reverse was found to be true only in a few cases. It is not unlikely that greater discrepancies might have been noted if roentgen examinations had been done earlier and more frequently.

The roentgen changes observed at the Boston City Hospital were essentially the same as those described by Schatzki, but some of them appeared to be more frequent and more extensive than those encountered at the Massachusetts General Hospital. In particular, the diffuse miliary type of mottling in large areas of the lung was seen more frequently in the early films. In some cases the individual lesions had a nodular appearance which varied considerably in density. The mottling extended throughout both lung fields in some of the severe cases, while in others it was limited to one or two lobes. Some patchy or confluent areas of density, probably those of bronchopneumonia, were also noted. This was particularly marked in the roentgenogram taken shortly before death in Case 107 as shown in Figure 11. Enlargement of the hilar shadows with increase in density and accentuation of the bronchial markings were quite frequent. In some cases, dilated and thickened bronchi could be seen extending down to the dome of the diaphragm.

Emphysema was evidenced by patchy areas of increased radiance, but in several cases this was extensive and predominantly unilateral and there was enlargement of the thorax on that side. Subcutaneous emphy-

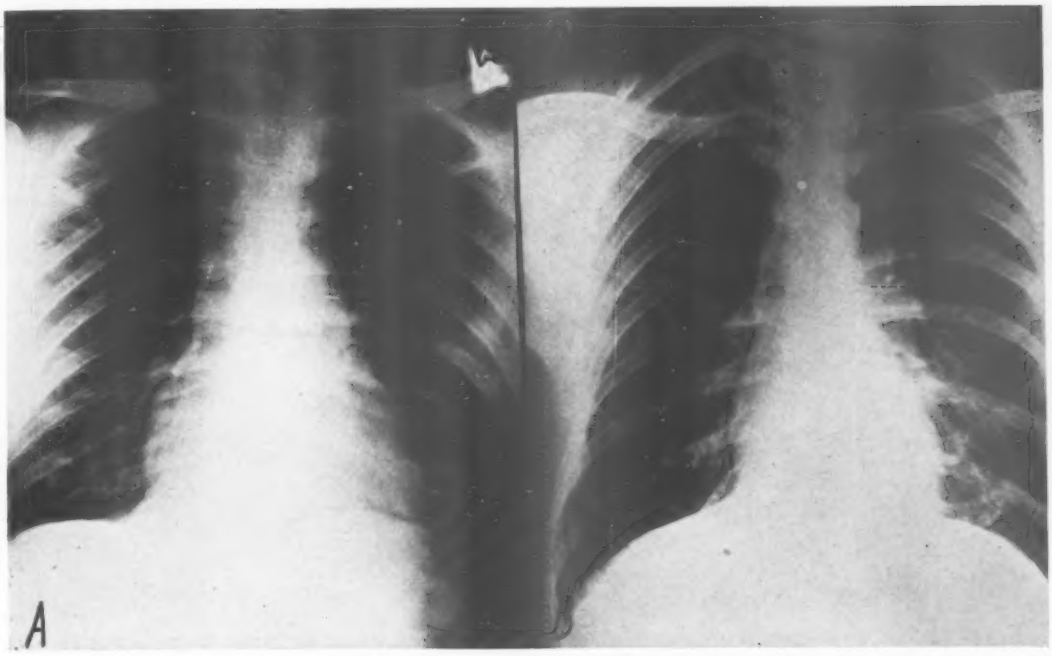

FIG. 5. Case 53. The fine areas of density, especially in the left lower lobe, which were present on the second day (*A*) have largely cleared and there is some increase in the bronchial markings on the seventeenth day (*B*).

The patient lost consciousness shortly after inhaling some of the smoke but he sustained only minor burns of the face. He had a chronic productive cough and a history of occasional asthmatic attacks. On admission he was coughing, his throat was diffusely inflamed and there were numerous râles throughout his lungs. His cough increased during the following day and he developed progressive dyspnea, restlessness and air hunger. The next morning there were signs of consolidation in the right lower lobe. These signs and symptoms cleared up dramatically during the day after a violent coughing spell following which the patient coughed up what looked like a fibrinous cast of a bronchus containing sheets of epithelial cells. The earlier roentgenogram was taken shortly after this episode and there were only crepitant râles heard at the bases at that time. There were occasional episodes of mild dyspnea during the next two days and the breath sounds were diminished at the lung bases on some examinations. Improvement continued and the lungs were clear and the patient symptom free when the later roentgenogram was taken. When last seen several weeks later he had remained well and without residual symptoms. Physical examination and roentgenogram of the lungs at that time were negative except for emphysema.

sema was noted in the roentgenograms taken after tracheotomy and this usually persisted for several days. It was limited mostly to the supraclavicular regions but in one instance extended into the axillae.

Atelectasis manifested by the various types of density described by Schatzki was seen in most of the positive roentgenograms. In addition, there were several cases in which the findings were interpreted as those of partial atelectasis of most of one lobe or one lung. The involved areas showed a more or less uniformly diminished radiance with elevation of the diaphragm, contraction of the intercostal spaces, and, in some instances, by an appreciable though not marked shift in the mediastinal contents to the affected side. Massive and complete atelectatic collapse involving most of the left lower lobe was noted in only 1 instance, Case 22, Figure 3, and in that case it persisted for several weeks. A large area of consolidation extending beyond the atelectasis appeared at the end of the fourth week in this case.

Pulmonary edema was not made out in any roentgenograms, but no postmortem roentgen examinations were made at this

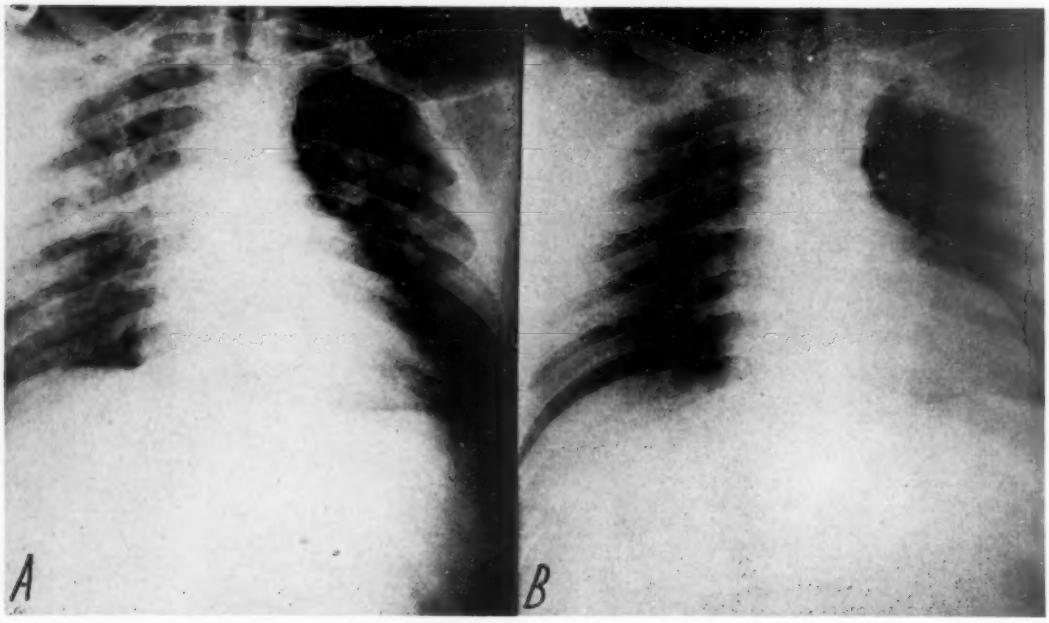

FIG. 6. Case 70. Diffuse irregular densities and some patchy emphysema on the second day (*A*); most of this has cleared by the sixth day (*B*) but there is some diminished aeration of the left lower lung.

The patient lost consciousness after inhaling much smoke. His surface burns were limited to the face. On admission he was stuporous, very hoarse, coughing vigorously and having some difficulty in breathing. At that time he was slightly cyanotic and coarse rhonchi were heard throughout both lungs and finer crepitant râles at the bases. During the next two days the patient was quite restless and dyspneic and he had noisy respirations with occasional stridor. He coughed violently and raised scanty amounts of dark mucoid sputum. At the time of the earlier roentgenogram there were fine râles at the lung bases and some scattered musical râles and the patient was having episodes of asthmatic-like breathing. After that the patient began to improve and the lungs gradually cleared but hoarseness persisted until discharge. When last seen two years later, the lungs were clear on physical and roentgen examination and he had remained free of respiratory symptoms.

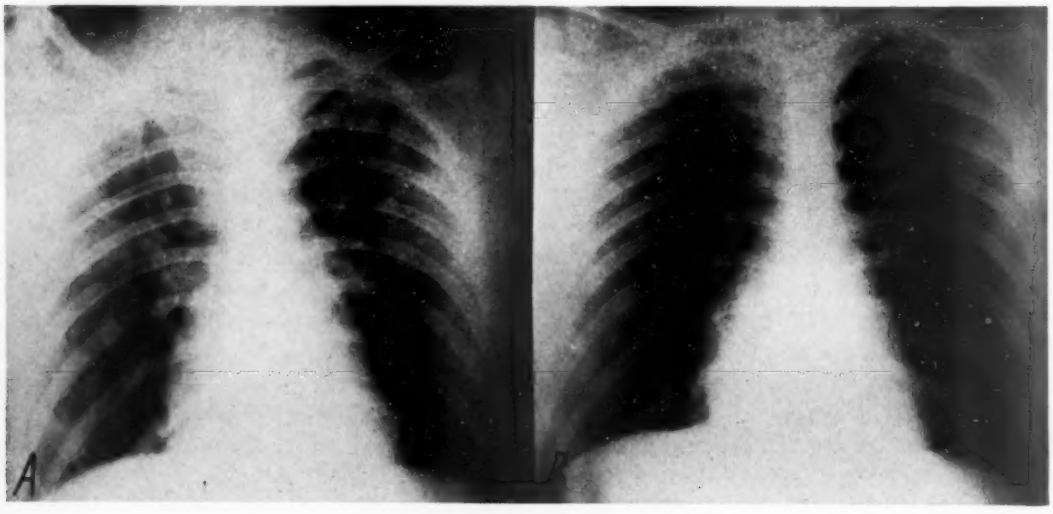

FIG. 7 (*See opposite page for legend.*)

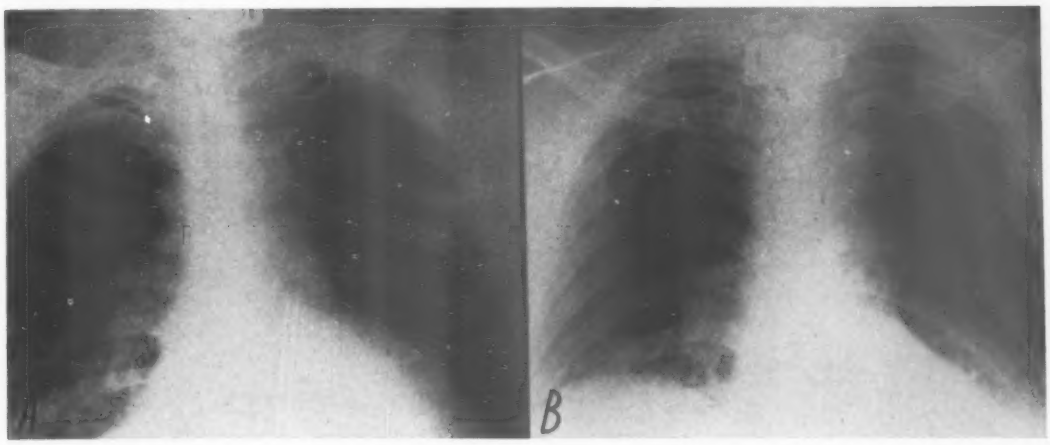

FIG. 8. Case 91. *A*, second day: tracheotomy tube in place; edema of soft parts and subcutaneous emphysema in supraclavicular and axillary regions; partial atelectasis of lower lobes with patchy densities in the right lower and emphysema in upper lung fields. *B*, eleventh day: tube still in place with similar but less marked changes.

The patient had temporary loss of consciousness before he got out of the burning building and again while being taken to the hospital. His burns involved the face and hands—about 6 per cent. There was a long history of morning cough productive of mucoid sputum. On admission he was hoarse, coughing violently and raising black, tenacious sputum. He soon developed increasing dyspnea and stridor and a tracheotomy was done on the following afternoon. Obstructive symptoms continued and necessitated frequent suction which yielded copious amounts of bloody mucoid material. The earlier roentgenogram was taken on the day after the operation when the lungs were filled with rhonchi and crepitant râles. Episodes of obstruction requiring suction continued for several days and the lung signs during that time were quite variable. When the later roentgenogram was taken there were only a few râles heard at the lung bases posteriorly and the breath sounds were diminished over these areas. The tube was removed a week later and the patient had a small hemoptysis on the following day. Thereafter, his symptoms improved rapidly. When last seen nearly two years after discharge he was in excellent health and had been carrying on his usual rather strenuous activities and having no more cough and sputum than he did before the fire. No abnormalities were made out on physical examination or roentgenogram of his chest at that time.

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FIG. 7. Case 90. *A*, second day: tracheotomy tube in place; mottled densities especially in upper lung fields; patchy atelectasis and emphysema. *B*, sixth day: tube out and lung fields cleared considerably. Right-sided cardiac enlargement—same in both roentgenograms.

The patient became unconscious while still in the Melody Lounge and, except for a brief interval soon after she was carried out through a window, she remained in this state until a few hours after her arrival at the hospital. There was a history of bronchial asthma but no recent attacks. Burns totalling about 5 per cent involved the face, a shoulder, a hand and a leg. The patient was irrational for several hours but dyspnea was first noted after about twelve hours and then progressed rapidly along with marked stridor until a tracheotomy was done. For several hours thereafter she continued to be restless and dyspneic with spells of breathlessness which were relieved by suction. When *A* was taken there were loud rhonchi and crepitant râles throughout both lungs. The patient began to improve rapidly, however, and breathed comfortably with the tracheotomy tube plugged, so it was removed a few hours later. Some dyspnea and wheezing continued and high pitched musical and some crepitant râles were heard in the lungs, mostly at the bases, for several days. During the next two years her general condition has remained good. She has some morning cough and sputum which occasionally is blood streaked but there have been no asthmatic attacks. Physical examination and roentgenograms of the lungs in inspiration and expiration were entirely normal.

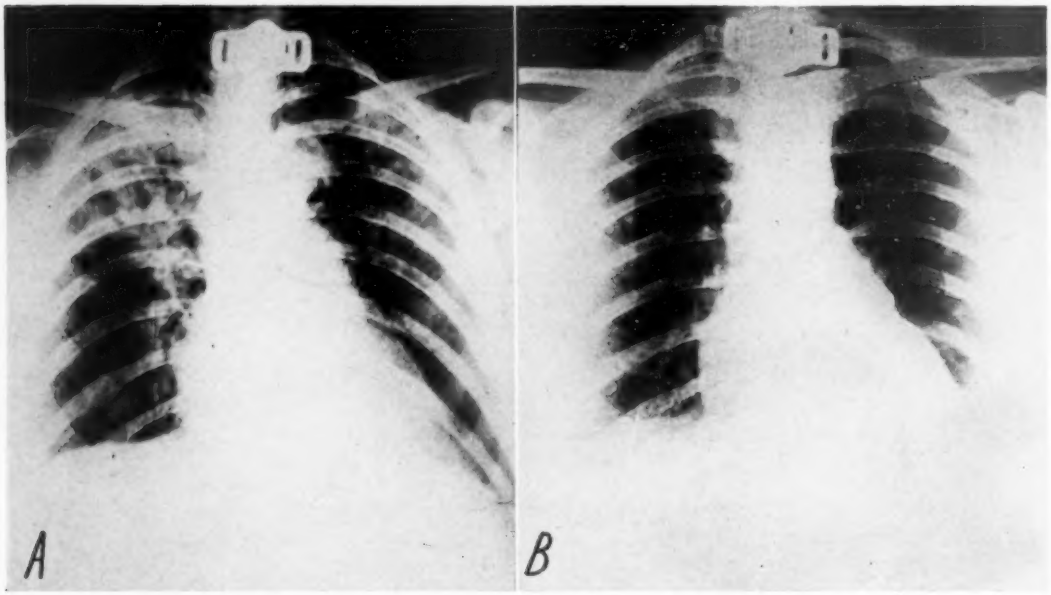

FIG. 9. Case 92. The tracheotomy tube is in place and subcutaneous emphysema in the supraclavicular areas are present in both roentgenograms; the marked densities seen on the second day (*A*) have largely cleared by the sixth day (*B*).

The patient inhaled a good deal of smoke and lost consciousness just before reaching an exit but she sustained no surface burns. On admission she had regained consciousness and was having some respiratory distress and numerous rhonchi and high pitched musical râles were heard throughout both lungs. During the next few hours her dyspnea increased markedly and she finally developed air hunger which necessitated a tracheotomy for relief. *A* was taken on the following day when the patient was still having considerable respiratory difficulty and there were numerous râles and rhonchi in both lungs at that time. She began to improve two days later and at the time of the later roentgenogram (*B*) was breathing much more comfortably but there were still many crepitant scattered musical râles. Except for a few choking spells due to plugging of the tracheal tube she improved steadily, and the tube was removed on the sixteenth day. Hoarseness, cough and a few rhonchi were still present when the patient was discharged one week later. When last heard from two years later she was entirely well except for a slight residual cough productive of very small amounts of mucoid sputum.

hospital in cases dead on arrival. This finding may also have been missed in fatal cases in which the last roentgenograms were taken some time before death. Extensive pulmonary edema, however, was not found, even in the autopsies of these cases. There was evidence of extensive and marked edema of soft parts, particularly of the face and neck in some of the cases as illustrated in Figure 14.

A summary of the numbers of cases in which each of the various types of roentgen changes were noted, and the relevant clinical findings are shown in Table II. As might be expected from the nature of the lesions, the clinical signs and symptoms were generally more frequent and more severe than was indicated by the roentgen changes. The discrepancies between the severity of the respiratory symptoms and roentgen changes in the lungs, on the one hand, and the extent of the surface burns, on the other, are seen from Table II and have been referred to previously.

In general, the more extensive roentgenographic lesions had largely cleared before the end of the first week. Small areas of atelectasis and patches of emphysema with some increases in the hilar shadows and of the bronchial markings persisted longer in some of the cases. The area of massive and complete atelectasis in Case 22 persisted unchanged for several weeks. In a few instances, there were marked fluctuations

TABLE II

SUMMARY OF RELEVANT ROENTGENOLOGICAL AND CLINICAL FINDINGS CLASSIFIED ACCORDING TO THE EXTENT OF THE ROENTGEN CHANGES SEEN IN THE LUNGS

Grade of Roentgen Changes		0	1	2	3	4	Total
Number of Cases		11	26	25	11	8	81*
Roentgen Findings							
Mottling:	coarse	—	1	6	8	7	22
	fine	—	18	18	2	1	39
	mostly unilateral	—	11	12	2	—	25
	bilateral	—	8	12	8	8	36
Emphysema:	patchy	—	14	19	9	5	47
	diffuse†	—	1	5	2	3	11
Atelectasis:	patchy	—	10	17	4	4	35
	diffuse† (partial)	—	15	8	6	4	33
	massive (complete)	—	—	—	1	—	1
Consolidation (mostly patchy)		—	—	—	3	2	5
Increased hila:	unilateral	—	9	21	—	—	30
	bilateral	—	11	2	11	8	32
Clinical Findings							
Symptoms:	hoarseness	3	15	24	10	7	49
	cough	6	22	25	11	8	72
	dyspnea	—	6	12	10	8	36
	wheezing	—	—	9	5	6	20
	stridor	—	—	9	7	7	23
Physical signs:	lungs clear	6	—	—	—	—	6
	crepitant râles	4	19	23	9	8	63
	musical râles	4	13	15	11	8	51
	diminished breathing	1	9	11	3	2	26
	consolidation	—	—	4	2	3	9
Extent of surface burns:							
0– 5%		6	16	12	3	3	40
6–10%		2	7	5	3	2	19
11–20%		2	0	2	1	1	6
21% or more		1	3	6	4	2	16
Deaths		1	2	2	5	5	15

* Six cases transferred to other hospitals within the first 36 hours are excluded from this table.
† Most of these had patchy areas as well.

in both physical and roentgen findings as might be expected. This was particularly striking in some of the cases with tracheotomy and in others who had repeated episodes of respiratory obstruction and required frequent suction to relieve their symptoms. Some of the roentgenograms in the 3 patients who recovered following tracheotomy, namely Cases 90, 91 and 92, are shown in Figures 7, 8 and 9, respectively. Most of the clearing in these cases had taken place within two or three days after the operation. The subcutaneous emphysema is also noted in these roentgenograms. Reproductions of roentgenograms taken in some of the other cases are also shown in the other figures and the relevant clinical findings are given in the accompanying legends.

The findings in some of the fatal cases

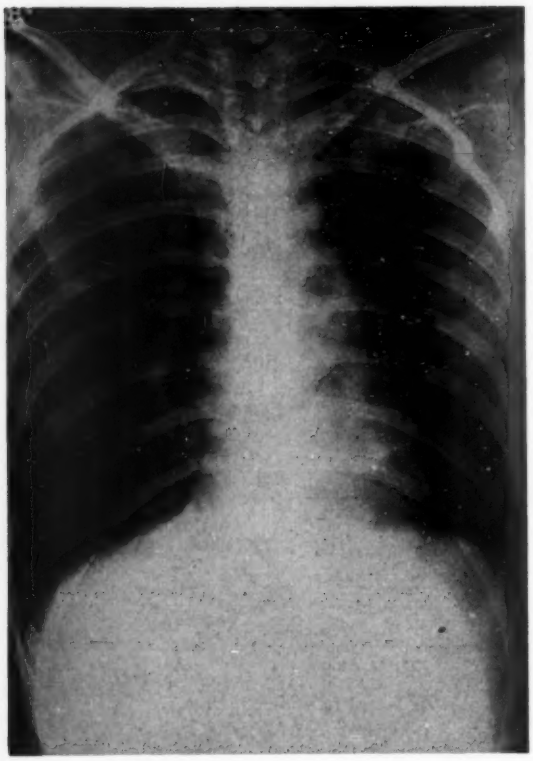

FIG. 10. Case 104. Second day: in both lung fields—more on the left—there are numerous areas of soft density but the extreme bases are clear.

The patient had apparently lost consciousness at the fire. She had burns of the face, arms and hands—total about 7 per cent. On admission, she was restless, excited and having respiratory difficulty, and coarse rhonchi were heard throughout both of her lungs. She continued to be restless and had episodes of excitement associated with stridor and air hunger. Showers of coarse crepitant râles were heard at both lung bases at the time of this roentgenogram. The patient obtained only slight and temporary relief from oxygen given under positive pressure. About thirty-six hours after this roentgenogram was taken the patient died during an episode of air hunger which was not relieved by tracheotomy. Just prior to the operation the lungs were hyperresonant throughout and only a few high pitched squeaks were heard.

At autopsy the trachea showed irregular areas of necrosis and some hemorrhagic foci. The mucosa of the larynx, trachea and bronchi was moderately injected and covered with a thick gray-black mucoid material. Some of this material also filled the smaller bronchi and was dislodged only with difficulty. The lungs showed scattered sunken bluish-red areas of consolidation within which there were obstructed bronchi and bronchioles. There was also some diffuse edema.

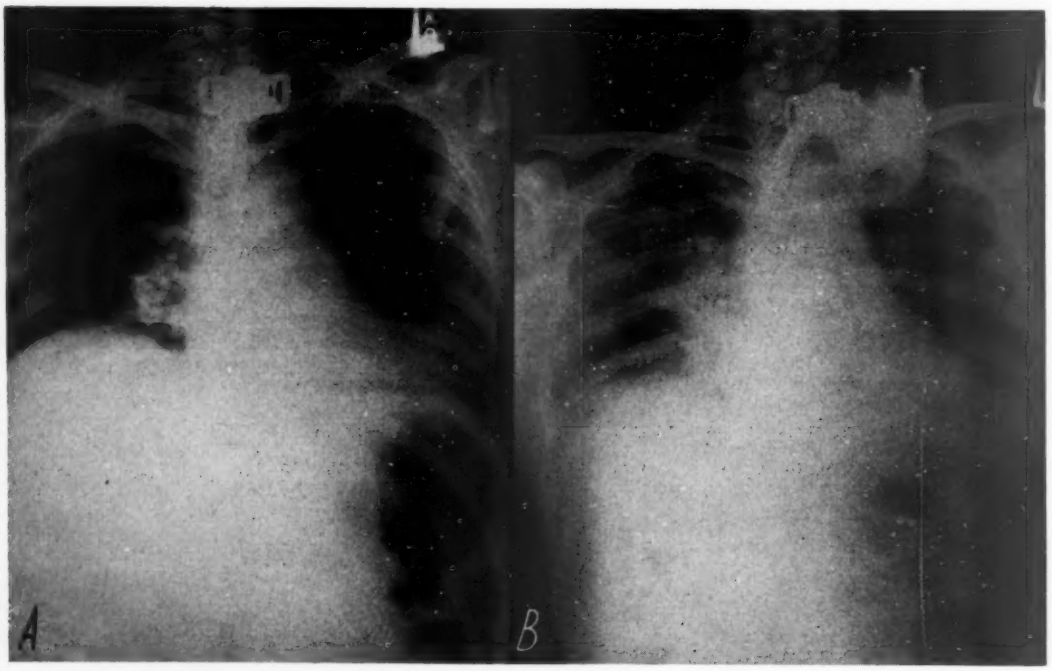

FIG. 11 (*See opposite page for legend.*)

Fig. 12. Case 124. Second day: extensive mottled densities and patchy atelectasis in the lower lung fields and emphysema in the uppers.

The patient lost consciousness while trying to escape from the fire and was stuporous and in shock on arriving at the hospital. He had severe burns of the face, neck and extremities—total about 10 per cent. On admission he was having spells of violent coughing and there were loud tracheal râles, a few scattered inspiratory wheezes and some fine crepitant râles at the bases. He developed marked air hunger and more profound shock after the first twelve hours. Suction was required frequently to relieve the obstruction which was partly due to moisture in the upper respiratory passages. The patient died eight hours after this roentgenogram was taken.

Autopsy showed edema and redness of the epiglottis, trachea and major bronchi which were covered with thick greenish and pinkish material but there was no underlying ulceration. In the smaller bronchi and bronchioles there were yellowish friable casts, and the underlying mucosa and part of the muscle walls were necrotic. There was a widespread early bronchopneumonia with polymorphonuclear cells and necrotic debris in the lumens of some of the bronchi and also some destruction of alveolar walls and early abscess formation.

are of interest. In Case 107 (Fig. 11), the first roentgenogram was taken after a tracheotomy had been performed and during a period when the patient was relatively free of obstructive symptoms. The lung fields in this roentgenogram were relatively clear. The patient had recurrent episodes of obstruction which were only partially and temporarily relieved by suction and she later developed signs of consolidation in the lungs. Roentgen examination at this time showed some confluent bronchopneumonia. An incidental finding was that of a megacolon which was noted in both roent-

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Fig. 11. Case 107. The tracheotomy tube is in place in both roentgenograms which also show the distended megacolon; many areas of marked density in the left cardiophrenic angle and some patchy atelectasis in the left lower lung on the second day (*A*) and more extensive and softer density especially in the right lung on the sixth day (*B*).

The patient was in shock and having some respiratory distress at the time of admission when rhonchi and wheezes were heard throughout her chest. She had deep burns on about 12 per cent of the body including her face, neck and extremities. Her breathing became increasingly difficult and a tracheotomy was done after eighteen hours. Thereafter, she had frequently recurring obstructive episodes requiring suction and this produced moderate amounts of viscid dark bloody material. She obtained some relief from oxygen under positive pressure after some of these episodes. At the time of the earlier roentgenogram there were numerous rhonchi and crepitant râles throughout both lungs. On the fifth day there were signs of consolidation in the left upper lobe and the râles persisted and increased in the rest of the lung. The patient died on the day after the later roentgenogram was taken.

At autopsy there was an opaque yellowish-gray pseudomembrane covered with dirty viscid mucoid material beginning in the larynx above the vocal cords and extending well into the primary bronchi. The smaller bronchi were dilated down to the diaphragmatic surface and there was some necrosis of their walls. Some bronchioles were filled with fibrin plugs. The lungs showed scattered areas of emphysema, atelectasis and consolidation and some confluent bronchopneumonia. The roentgen finding of a megacolon was confirmed.

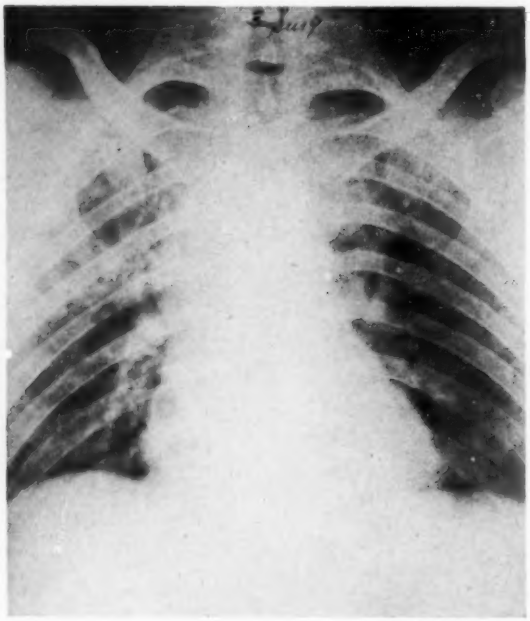

FIG. 13. Case 125. Second day: diffuse haziness of most of both lung fields with scattered areas of density of various shape, particularly in the lower portions; widened right supracardiac area.

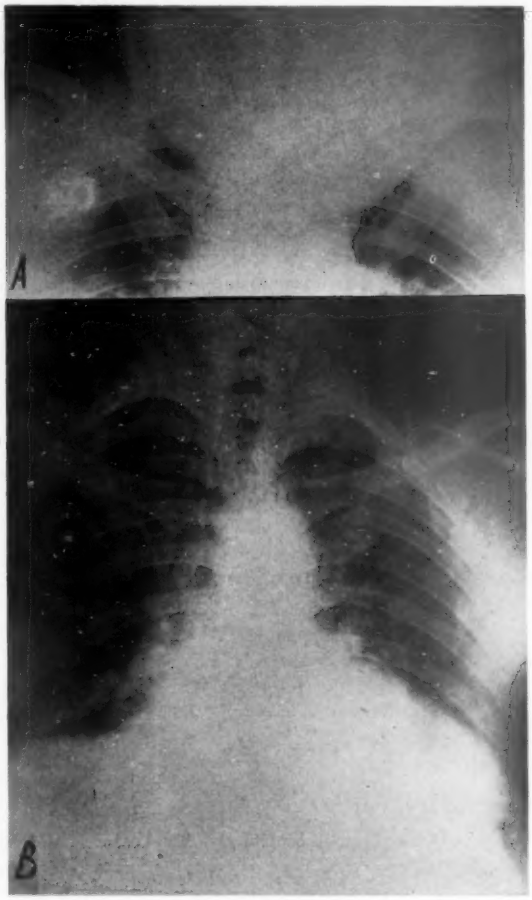

FIG. 14. Case 6. *A*, taken on the second day, shows the marked swelling of the face and neck which had almost entirely receded when *B* was taken on the sixth day.

At the time of admission the patient was semicomatose, in shock, hoarse, coughing and having some difficulty in breathing, and coarse râles were heard throughout both lungs. Burns were severe and extensive and involved about 45 per cent of the body, including the face. During the first twelve hours respirations became increasingly noisy and labored and dark mucus was removed by suction. Black and later bloody mucus was raised by the patient or removed by suction frequently thereafter. There were scattered râles at the lung bases posteriorly at the time this roentgenogram was taken but breath sounds over these areas were subsequently diminished. Dyspnea persisted and progressed and the patient died on the sixth day.

At autopsy there was a soft edema of the lower pharynx and small submucosal hemorrhages and hyperemia of the mucosa of the epiglottis, larynx, trachea and bronchi which were covered with a film of thin mucopurulent material. The lungs contained multiple petechial and large confluent hemorrhages over the posterior and lateral surfaces and many areas of superficial atelectasis. There was somewhat generalized emphysema of the upper lobes and a large infarcted area in the left lower lobe with other scattered areas of infarction, atelectasis and trapped air as well as fibrin thrombi filling many small bronchi and bronchioles the walls of which showed considerable necrosis.

genograms. These findings were confirmed at autopsy and the characteristic severe laryngitis, tracheitis and bronchitis with areas of atelectasis and emphysema were also present.

In Case 104 (Fig. 10) the roentgenogram was taken about thirty-six hours before death, which resulted from acute respiratory obstruction that was not relieved by tracheotomy. Examination of the lungs just prior to the operation showed them to be hyperresonant throughout and only high pitched musical sounds were heard. Autopsy in this case showed acute tracheitis,

laryngitis, bronchitis and bronchiolitis with surrounding small foci in the alveoli. There was some rather diffuse atelectasis and edema.

In Case 128 there were no significant roentgen findings. The patient had extensive burns and died during the twelfth week. His lungs were entirely clear at autopsy. In a second patient with extensive burns (Case 109) there were only small areas of atelectasis and emphysema and some increase in the hilar shadow in the roentgenograms taken during the first week. The patient had only mild and transient dyspnea and a slight cough. No definite abnormal physical findings were made out in her chest. The patient died after five months and the lungs were found to be entirely normal at autopsy.

In Cases 106 and 120, moderate changes (Grade 2) were made out in the roentgenograms taken on the second day. The former died about twelve hours later during a sudden attack of acute obstruction which was not relieved by tracheotomy. Her lungs were resonant throughout and only a few high pitched râles were heard at the time. There was no autopsy in this case. In Case 120, death occurred about thirty-six hours after the roentgenogram was taken. Numerous crepitant râles were heard throughout the lungs in this case. Autopsy showed hemorrhagic acute laryngitis, tracheitis and bronchitis with focal areas of hemorrhagic consolidation in the lungs. The remaining fatal cases all had more extensive roentgenographic changes and all showed patchy areas of atelectasis and emphysema in addition to the lesions in the larynx, trachea and bronchi. There were some areas of bronchopneumonia in those dying after the fourth day and these areas were confluent and almost lobar in distribution only in Case 107.

Follow-up roentgen examinations in inspiration and expiration were done in many of the patients who had moderate or extensive changes in the early roentgenograms. No residual abnormalities were detected in roentgenograms of any of these cases six months to two years after the fire.

SUMMARY

The significant roentgenographic changes observed in the cases admitted to the Boston City Hospital from the Cocoanut Grove fire have been reviewed and related to the clinical and autopsy findings. The roentgen changes were essentially similar to those described by Schatzki and were consistent with the lesions found at autopsy. Most of them could be ascribed to atelectasis and emphysema resulting from a severe membranous laryngotracheobronchitis with obstruction. The clinical symptoms and physical signs were frequently more severe and extensive than the changes visible in the roentgenograms. No residual roentgen changes in the lungs were noted in the recovered cases in which there was an opportunity for follow-up.

The authors are indebted to Dr. Patrick F. Butler, Director of the X-ray Department, for his constant interest and encouragement, to Drs. C. C. Lund and F. H. L. Taylor, the responsible investigators of the Burns Project, to the members of the surgical staff of the Boston City Hospital who permitted us to study their cases and to Dr. Timothy Leary for the pathological data.

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PULMONARY CHANGES IN CARBON TETRACHLORIDE POISONING*

By LIEUTENANT (JG) CHARLES MOREAU THOMPSON (MC) USNR

RECENTLY a series of 20 cases of carbon tetrachloride poisoning occurring on a submarine were treated at this Naval Dispensary. Of the cases, 3 were critically ill and 1 died. During their illness roentgenograms of the chest were made. Some very unusual changes were noted that are deemed worthy of reporting. It was impossible to make a complete survey of the literature at this outlying base, but in none of the papers reviewed was mention made of roentgen changes in the lung fields.

Carbon tetrachloride is an excellent solvent for grease, fat, tar and oils, and used not uncommonly for cleaning purposes. Aboard the submarine the solvent was used in a confined compartment for cleaning the deck and bulkheads. When exposed to high temperature carbon tetrachloride can break up into phosgene and hydrochloric acid, both of which are pulmonary irritants. However, it is the opinion of the author that the men were poisoned by the direct inhalation of the fumes. The metal plates being cleaned were not hot, and none of the men initially had any symptoms of phosgene inhalation. The pulmonary changes were not discovered until nine days after exposure.

CLINICAL MANIFESTATIONS

The course of the cases reported below is similar to that in the literature except for a few variations. Of the 20 cases, 4 were severely ill, presenting a typical toxic nephrotic syndrome as evidenced by puffiness and swelling of the soft tissues of the face, hands, and feet. They had retention of nitrogenous waste products in the blood stream and evidence of real kidney damage. Due to limited laboratory facilities, complete blood and urine studies were not possible. The fatal case developed acute pulmonary edema while apparently on the road to recovery. Because of this, roentgenograms were made of the chest of each patient. Findings varying from mild prominence of all lung markings to complete consolidation of all five lobes were observed. Enlarged hilar shadows, peribronchial infiltration and a change in the size and configuration of the heart were also noted. The patient with pulmonary symptoms had extensive consolidation of both lung fields and died despite all supportive treatment.

CASE REPORTS

The 4 serious cases are outlined in brief, along with the gross and microscopic findings of the lungs in the fatal case.

CASE 1. P. DeS., male, white, aged twenty-four. Patient admitted with backache, malaise and vomiting. He had severe oliguria with four plus albuminuria. For six days he was acutely ill at which time he appeared to be improving and seemed to be the least ill of the critical patients. Suddenly the patient developed acute respiratory distress with deep cyanosis and hemoptysis. Respirations were 34 per minute with a pulse rate of 120 per minute. Roentgen examination of the chest was made. Temperature was normal and remained so. Course râles and wheezes were heard over both lungs. The patient became rapidly worse, respirations reached 40 per minute and pulse 140 per minute. Because of the marked cyanosis and air hunger he was placed in an oxygen tent. He began to cough up large amounts of red frothy sputum. Patient died about thirty hours after the onset of his pulmonary symptoms.

Autopsy and Microscopic† Findings of the Lungs. Gross:* There were petechial hemorrhages on the smooth glistening surfaces of the lungs. The lungs were heavy and, with the

* Autopsy performed by Captain H. Salm, M.C., A.U.S.

† Microscopic examination performed by Colonel F. H. Foucar, M.C., A.U.S.

* The opinions or assertions contained herein are the private ones of the writer and are not to be construed as official or as reflecting the views of the Navy Department or the Naval Service at large.

exception of parts of the lower lobes, were considerably consolidated. The cut surfaces of each were dark red and very wet. The mucosa of the tracheobronchial tree was a dark purplish red. *Microscopic:* There was marked venous capillary congestion and edema. The alveoli were filled with granulocytes, numerous erythrocytes and many swollen lightly pigmented macrophages. Findings—congestion and edema; pneumonia, acute, hemorrhagic in type.

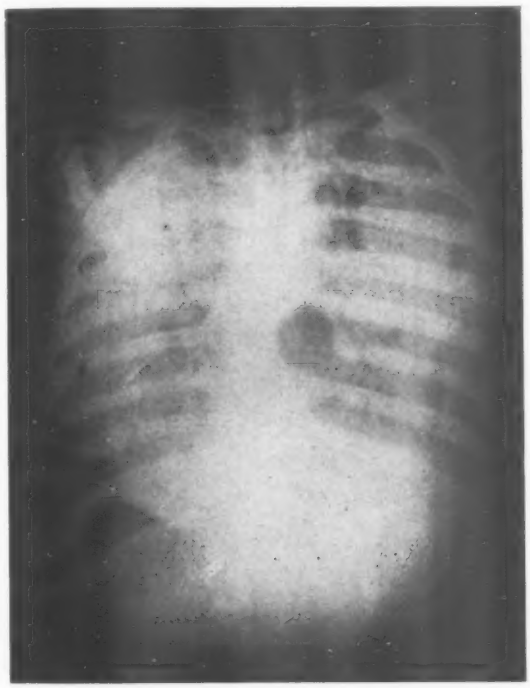

FIG. 1. Case I. Roentgenogram of the chest reveals a homogeneous density involving the lung fields. There is only a small amount of aerated lung at the right apex and at the left base. The cardiac silhouette is obscured by the consolidation in the lung fields. There is no definite evidence of free fluid in either pleural space. (This is not an underexposed film.)

CASE II. A. B., male, white, aged twenty-three, admitted with nausea, vomiting, albuminuria, and oliguria. Albuminuria persisted for fourteen days. Shortly after admission facies became bloated, edematous and waxy. He was critically ill for two weeks with nausea, vomiting with hematemesis, oliguria and anorexia. He was fed intravenously and convalescence was slow. Roentgen examination of the chest was first made on the seventh hospital day. In spite of roentgen evidence there were no physical or clinical findings in the chest. Two days later a roentgenogram of the chest was essentially negative. Patient was discharged on the thirty-second hospital day.

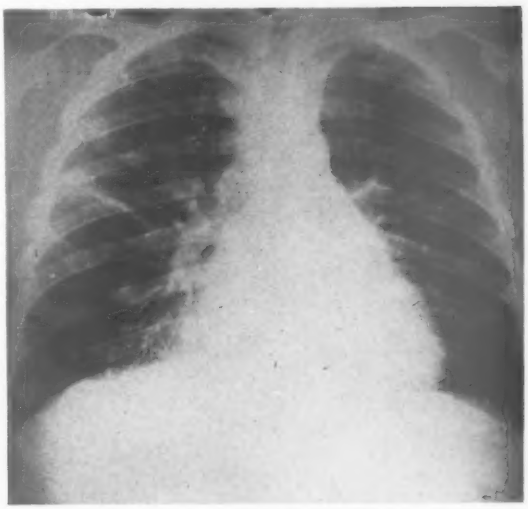

FIG. 2. Case II. Roentgenogram of the chest reveals an increase in the hilar and lung markings. The markings fan out into the periphery of the lung fields. In the mid-portion of the right lung field there is a linear line of increased density probably due to interlobar pleural thickening or focal atelectasis. The cardiac silhouette is slightly globular in shape.

FIG. 3. Case II. Re-examination of the chest three days later reveals marked resolution of the prominent lung markings previously described.

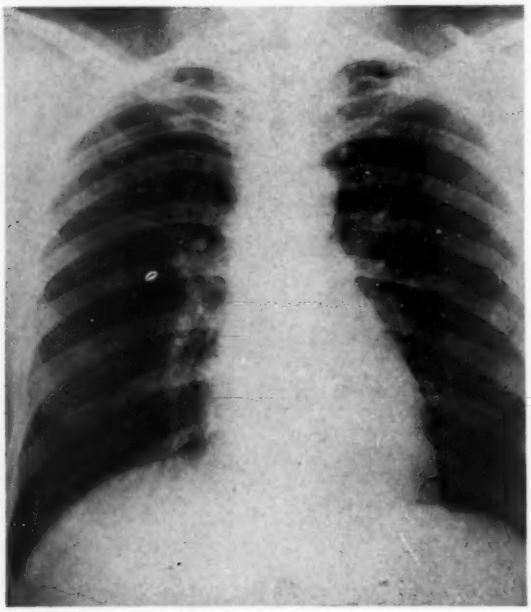

FIG. 4. Case III. Roentgenogram of the chest reveals an increase in the prominence of the hilar markings and root shadows. The cardiac silhouette appears normal. Re-examination three days later revealed the lung fields to be normal.

CASE III. J. B., male, white, aged twenty-two. Patient admitted with severe vomiting and low back pain. Oliguria and albuminuria were severe and persisted for a week. Shortly after admission he developed large flame-like

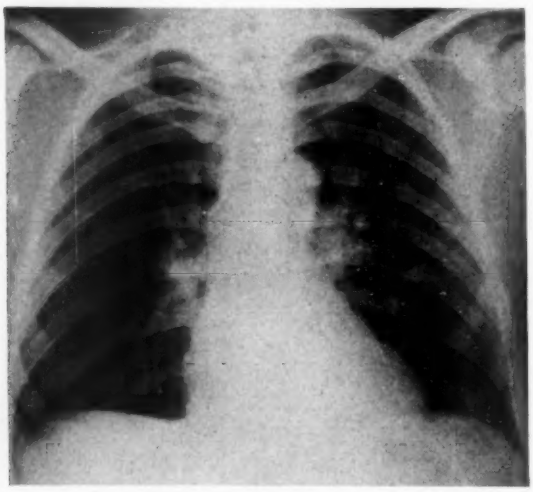

FIG. 5. Case IV. Roentgenogram of the chest reveals prominence, enlargement and increased density in both hilar areas with fine linear shadows radiating out into the lung fields.

subconjunctival hemorrhages. Roentgenogram of the chest was made on the seventh hospital day. He was the least ill of the 3 living patients. Discharged on thirty-second hospital day.

CASE IV. L. J., male, white, aged twenty-four, admitted with severe prostration, vomiting and generalized muscular pain. There was anuria for five days followed by oliguria and albuminuria. He developed subconjunctival hemorrhages and marked photophobia on the

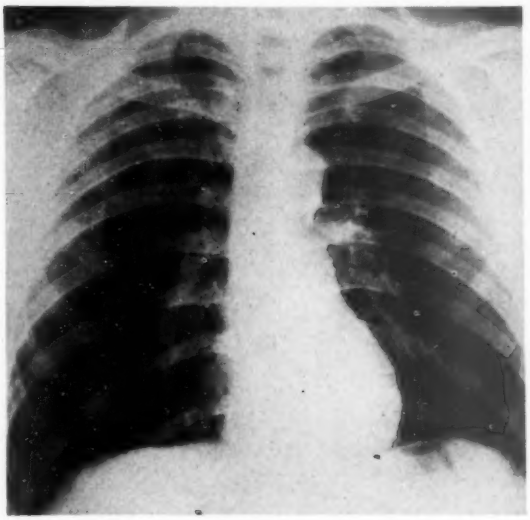

FIG. 6. Case IV. Re-examination of the chest one week later reveals marked improvement of the hilar and lung infiltration previously described. The lung fields now appear normal. It is noted that there has been marked change in the configuration of the cardiac silhouette. The heart on this examination is not as globular in shape. Part of this change in the cardiac silhouette may be attributed to the greater descent of the diaphragm and better aeration of the lungs.

second hospital day. The soft tissues of the face became edematous and waxy. He had nitrogenous retention. The patient was unable to tolerate food by mouth for two weeks and required intravenous feeding. Roentgenogram of the chest on the seventh hospital day showed marked prominence and infiltration of both hilar areas. A second examination one week later revealed a decrease in the pulmonary infiltration and a change in the configuration of the cardiac silhouette. Convalescence was slow and he was discharged on the thirty-fourth hospital day after the most stormy course of the living patients.

SUMMARY

Four in a series of twenty cases of poisoning by inhalation of carbon tetrachloride fumes have been presented along with roentgenograms of the chest. The clinical course of each case and the autopsy findings in the fatal case were consistent with those described in the literature. Pulmonary roentgenographic changes were discovered late in the course of events. The changes varied from consolidation of all five lobes to mild increase in all lung markings. In one case there was a change in the configuration of the cardiac silhouette between examinations. Only the fatal case had symptoms and physical findings of organic change in the lung fields. In the other three patients the predominating symptoms were gastrointestinal, followed by renal shut-down.

It is interesting to note that the amount of change on the roentgenograms of the chest was directly proportional to the severity of the patients' clinical illness; i.e., the more ill the patient, the greater were the findings on the roentgenogram. Perhaps pulmonary roentgen changes may be used as criteria in the prognosis and morbidity of cases with carbon tetrachloride poisoning.

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ESOPHAGOBRONCHIAL FISTULA THROUGH AN ESOPHAGEAL DIVERTICULUM

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ESOPHAGOBRONCHIAL fistulae are not exceedingly rare but the occurrence of such a fistula through an esophageal diverticulum has apparently been reported only once. It is for this reason and for the therapeutic possibilities of the cauterization method of Clerf[3] in the treatment of bronchoesophageal fistulae that our case is presented.

CASE REPORT

Mrs. J. M. (admission No. 114114), female, married, white, aged forty, entered Mount Sinai Hospital on September 21, 1941, with the chief complaint of "hemorrhage through the nose and mouth." She stated that such an episode first occurred one afternoon in 1940 while hurrying to board a streetcar, the blood loss being about one-half pint. When the patient returned home, she felt faint, nervous, restless and dyspneic. The bleeding gradually subsided but less severe attacks of bright red bleeding occurred subsequently.

FIG. 1. Spot roentgenogram taken during barium meal examination on September 22, 1941, shows a tract (*T*), probably fistulous, extending posteriorly and inferiorly into the right lung region from diverticulum (*D*) in lower half of esophagus.

The patient awoke during the night of September 9, 1941, and regurgitated about a "pint" of dark clotted "chunks" of blood, and on the following day, she vomited bright red blood. She also complained of severe epigastric, right shoulder, and neck pains, the complaints being intensified while eating. She noticed that there was an increase in eructations, but they were not of the sour type. The patient claimed she was able to drink milk but could not tolerate fried or greasy foods or cream. The stools appeared darker than usual but not black.

The past history included "flu" in 1918, a hysterectomy in 1932 and a ventral hernia repair in 1940. The patient had 5 miscarriages and 5 living children, having married at the age of fifteen.

Physical Examination. On entrance to the hospital this obese, white, pale female was in acute pain. There was marked right upper abdominal tenderness which was increased during deep inspiration but no rigidity or spasm was found. The liver, kidneys and spleen were not palpable. An incisional hernia was recognized in the lower abdomen. No definite abnormal physical findings were elicited in the heart or lungs. The blood pressure was 92/60, pulse 84. The impression on admission was (1) bleeding gastric ulcer, (2) chronic cholecystitis with cholelithiasis.

Laboratory Data. The admission red blood count was 4,250,000 per cu. mm.; hemoglobin 13 grams; white blood cell count 11,800 with a normal differential count. The Kline test was reported as negative. A catheterized urine specimen showed no definite abnormalities.

Roentgen examination of the chest on September 22, 1941, by means of roentgenoscopy showed no gross abnormality in the heart, lungs, mediastinum or diaphragm. Upon administering a thick barium sulfate mixture,

the esophagus was opacified and a diverticulum measuring approximately 3 cm. in diameter was visualized in the lower half. A tract leading posteriorly from the diverticulum for about a distance of 1 inch was observed (Fig. 1). Whether there had been a communication between the esophagus and bronchial tree was not stated at that time, but review of the roentgenograms, however, leads one to believe that there must have been a communication. In the region of the diverticulum considerable spasm was noted. There was no evidence of neoplastic involvement. The stomach and duodenum showed no abnormality.

Course. On September 23, 1941, two days after admission, esophagoscopy was performed by Dr. John D. Steele and revealed the following: "At a point approximately 10 cm. above the cardia, considerable thin, purulent secretions were seen coming from the right wall of the esophagus. After aspiration of these secretions, the diverticulum was easily seen. The opening of the diverticulum was approximately 1 cm. in diameter. The diverticulum was approximately the size of a small olive and was lined with normal mucosa. The orifice of the fistulous tract was easily seen in the distal wall of the diverticulum. This orifice was circular and was approximately 3 mm. in diameter. There was no evidence of malignant change at any point."

Treatment consisted of bedrest, sedation, antispasmodics and a bland ulcer diet. The patient was discharged after a hospital stay of only three days.

Follow-Up Record. Except for occasional episodes of slight bleeding, the patient's general condition was good until July 10, 1942, when she again had a massive hemorrhage and was taken to the Milwaukee County Emergency Hospital. One week later she was discharged.

This patient was readmitted to Mount Sinai Hospital on January 26, 1943, with a six day history of chills, fever, cough, hemoptysis, emesis, and pain in the interscapular, left anterior chest and epigastric regions. In addition, she complained of burning on urination and noticed foul smelling urine. The patient appeared acutely ill and was expectorating thick, tenacious, bloody sputum. Examination of the heart was essentially negative and the blood pressure was 130/80. The pulse was rapid and weak, the rate being 120. Percussion of the chest revealed dullness in the left base.

Fig. 2. Esophagram made September 2, 1944, shows the esophagobronchial fistula. The fistulous tract is demonstrated between the esophageal diverticulum (*D*) and the bronchial tree (*B*).

Bronchial breathing and moist, loud, crackling râles were heard in this area but wheezes were perceived over both lung fields. Tenderness was elicited in the right upper quadrant of the abdomen but no abdominal masses were palpable. Some fever was present, the temperature on admission being 101.2° F. The diagnosis was lobar pneumonia. Chemotherapy in the form of sulfathiazole was administered and the temperature fell to normal on the second day. The temperature remained normal thereafter and the patient was discharged after a hospital stay of nine days.

About one and a half years later, or on August 25, 1944, the patient entered the hospital after she developed rather acutely, dyspnea and cough, followed by episodes of regurgitation and abdominal distention. The patient appeared slightly cyanotic, somewhat clammy and was distended. Percussion of the heart revealed suggestive enlargement to the left. The heart tones seemed weak and distant but no murmurs were heard. The blood pressure reading was 166/90, pulse was 94, and the temperature was 98.2° F. The abdomen, chiefly the left upper quadrant, was distended and presented a tympanitic note to percussion.

The impression gained was an acute gastrectasis. An electrocardiagram showed no evidence of myocardial involvement. The sedimentation rate was normal and the white blood cell count

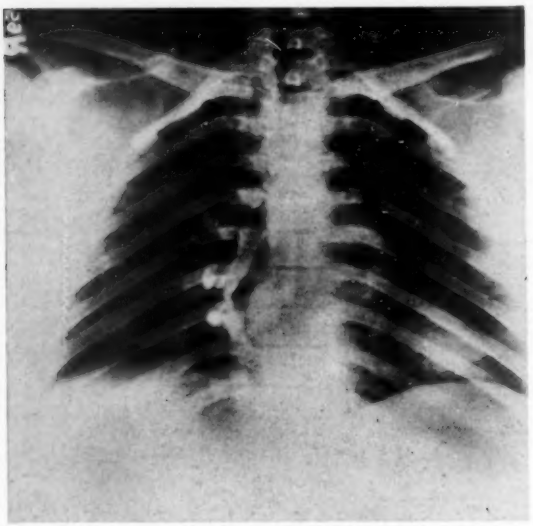

FIG. 3. Posteroanterior projection after ingestion of barium meal (September 2, 1944) shows filling of esophageal diverticulum and right main bronchus.

was 8,200. Morphine was administered and Wangensteen suction was instituted. Within twenty-four hours the distention had disappeared and the patient was free of symptoms. On the third day the patient was dismissed.

Roentgen examination of the esophagus and chest as a clinic patient on September 2, 1944, revealed the following: A diverticulum of the esophagus was opacified by the barium mixture and measured 3.5 cm. in diameter (Fig. 2). The diverticulum was situated posteriorly and approximately in the middle of the lower half of the esophagus. A fistulous tract was visualized which extended from the posteroinferior aspect of the esophageal diverticulum to a right lower lobe bronchus. The right main bronchus was filled shortly thereafter (Fig. 3) and the patient proceeded to expectorate the opaque medium. The right lower bronchi showed some saccular dilatation.

Preliminary examination of the chest showed prominence of the lower portion of the right hilar shadow. A few bronchopneumonic areas were also noted in the right lower lung medially.

In view of the usual poor outlook for such a patient, it was agreed that some therapeutic measure or measures would have to be instituted in an attempt to seal off the communication between the esophagus and bronchial tree. The patient was therefore readmitted to the hospital on October 24, 1944, for esophagoscopy and cauterization of the fistulous tract. This was attempted according to the method of Clerf and was performed by Dr. John D. Steele as follows: "The 9 by 45 esophagoscope was easily passed into the esophagus through the right pyriform sinus. The esophagoscope was first advanced to the cardia and then brought back along the right lateral wall of the esophagus until the diverticulum was encountered, as previously approximately 10. cm. above the cardia. The opening of the diverticulum into the esophagus was approximately 1.5 cm. in diameter. The diverticulum tapered down over a distance of approximately 2 cm. At the distal end of the diverticulum a circular opening approximately 3 mm. in diameter was present (Fig. 4). The mucosal lining of the diverticulum was normal in appearance. On inserting the aspirator through the opening at the end of the diverticulum the patient coughed violently, undoubtedly indicating that the aspirator had entered the bronchus. The aspirator could not be passed for more than a distance of 0.5 cm. beyond the visible portion of the opening. A small amount of bleeding followed passage of

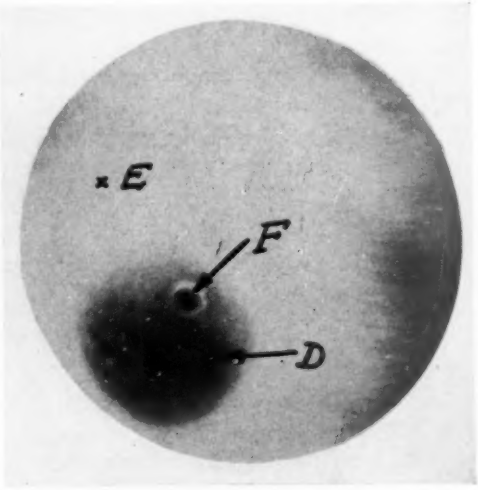

FIG. 4. Drawing of the esophagoscopic findings of Dr. John B. Steele on October 24, 1944. The esophageal diverticulum (*D*) is represented by the darkly shaded area and the fistulous opening (*F*) is demonstrated by the ring-like area. The lightly shaded portion is the wall of the esophagus (*E*).

the aspirator through this opening. The end of an applicator to which fused sodium hydroxide had been applied was then introduced into the mid-portion of the diverticulum and allowed to remain in place for exactly two minutes. On withdrawal of the applicator it was noted that considerable reaction had taken place, the mucosa lining the entire diverticulum now being bluish black in color and somewhat edematous. A swab soaked in boric acid solution was applied to this area. A small stomach tube was then inserted into the stomach through the nose."

Tube feeding was carried on for the following two days but the patient could not tolerate the stomach tube any longer. A soft diet was prescribed on the third postoperative day. The patient was discharged on November 2, 1944, but she had many episodes of hemoptysis in the interval. The patient was quite weak but showed no evidence of secondary anemia.

Esophagram on December 12, 1944 (Fig. 5) still showed the esophagobronchial fistula but the diverticulum was approximately one-half the original size. The patient has to date refused further treatment.

Comment. One can only postulate or theorize on the formation of the fistula in our patient. We believe it is fair to assume that the fistula was due to a ruptured gangrenous diverticulitis. Mediastinitis, pleuritis, pneumonitis and abscess formation probably followed. Rupture of the abscess into the bronchus then resulted in the esophagobronchial fistula.

The apparent failure of the one-stage cauterization in our case might be due to one or more factors. Perhaps the diameter of the communication or tract was too great to expect complete closure after just one cauterization. We believe that failure of complete closure of the tract was, at least in part, due to the fact that the patient pulled the stomach tube out after two days. The patient refused to have the tube reinserted.

DISCUSSION

Lahey[6] states that the presence of an esophageal diverticulum was first recognized in 1764 by Ludlow at postmortem examination. Diverticula of the esophagus are ordinarily classified into pulsion and traction types. The pulsion diverticula are divided further into the pharyngo-esophageal and supradiaphragmatic forms. The pharyngo-esophageal diverticulum is attributed to a congenital weakness of the wall between the oblique and transverse fibers of the cricopharyngeus muscles. Congenital weakness of the muscle fibers is also said to be the cause of the supradiaphragmatic or "epiphrenial" diverticula. Diverticula occurring below the tracheal bifurcation are usually in the middle of the lower half of

Fig. 5. December 12, 1944. Esophagram shows reduction in size of esophageal diverticulum after cauterization therapy on October 24, 1944. The fistulous tract is evidently narrower.

the esophagus.[10] The pulsion diverticulum is more common in males than in females. The supradiaphragmatic type of pulsion diverticulum is rare, Lahey reporting only 4 cases up through the year 1940.

The traction diverticulum results from an inflammatory process in the adjacent tracheobronchial lymph nodes or mediastinal pleura with the production of cicatrization which tends to pull the esophagus out in a lateral or anterior direction. These occur chiefly at the level of the tracheal bifurcation. When the pulsion factor is added to traction diverticula, the latter become larger and rounded. Early in the develop-

ment of the traction diverticula, there is supposedly no muscular atrophy.

As with esophageal diverticula, the literature contains numerous reports of esophagobronchial fistulae. In most instances these fistulae have resulted from cancer of the esophagus, tuberculous periadenitis, syphilis of the esophagus, a congenital abnormality or a foreign body in the esophagus.[7] By far the most frequent etiologic factor is esophageal cancer, and significant reports include those by Clerf[3], Clerf, Cooley and O'Keefe,[4] Drew and Ormerod,[5] Allen,[1] and Brown.[2] In 1930 Sigora[9] reported a case of esophageal diverticulum which perforated into the pleural cavity with the development of an empyema. The cause for the traction diverticulum was not known.

Pape[8] in 1934 presented a case report of a chronic esophagobronchial fistula through an esophageal diverticulum. This patient had two large and two small traction diverticula with a lower small one perforating into a right lower bronchus. The right lower lobe in this case showed signs of an old healed tuberculosis with evidence of calcification. This patient was a sixty year old white male who for two years complained of pain in the upper abdomen which was transmitted to the right chest. Other symptoms included acid belches, nausea and occasional emesis of tasteless liquids. The therapy instituted consisted of advising the patient to lie on the side opposite to that of the diverticulum in order to impair the passage of food particles into the sac. The patient was also advised to eat thick pasty foods which would not pass into the diverticulum as easily as fluids might. Eighteen months after instituting this regimen, there was noted a decrease in complaints with a narrowing of the fistulous tract as well as an associated weight gain.

Diverticula of the esophagus are usually incidental findings during roentgen examination of the gastrointestinal tract. Unless an inflammatory process is present, they probably do not give rise to any symptoms. The first complaint in a pulsion diverticulum is usually a disturbance in swallowing. Pulsion diverticula present symptoms which are related to food decomposition. Due to stasis in the sac over a long period of time, the patient regurgitates the contents of the diverticulum during the night or with change in position from the erect to the recumbent. In the case of traction diverticula, there may be no symptoms because the orifice is supposedly in a more dependent position than the sac and there is no problem of emptying. Even in the recumbent position there should be little if any difficulty. Traction diverticula, at least early in their development, contain a muscular coat and are therefore capable of contraction. Contents are therefore less likely to stagnate in such diverticula. Symptoms may accompany even such diverticula if active inflammatory changes are present. Partial obstruction due to spasm will give rise to difficulty in swallowing and even pain.

The symptoms of a simple esophagobronchial fistula consist essentially of inability to swallow food or liquid without initiating a violent episode of coughing or choking. There may be expectoration of sputum mixed with food. Hematemesis and hemoptysis can also occur. Retrosternal pain might be caused or aggravated by food intake. Interestingly enough, pain in the upper abdomen radiating to the right chest or shoulder occurred in Pape's case and in our patient. In the latter this pain was overshadowed by the hemorrhages. Nevertheless, this complaint was of sufficient concern for the examining physician to consider gallbladder disease.

ROENTGEN ASPECTS

Diverticula of the esophagus are in most instances first discovered by means of roentgen examination. The roentgen examination is often not very simple, especially if there is an associated inflammatory process. In any event, it is essential to examine the patient in all positions, including the

recumbent and even Trendelenburg position. Often it is necessary to vary the viscosity of the barium sulfate mixture in order to visualize the diverticulum. Other factors which might prevent filling of the diverticulum are debris in the diverticulum, occlusion of the neck of the diverticulum by an inflammatory process, and so forth.

The pure traction diverticulum, before the pulsion factor is added, will probably not be visualized unless the patient is examined in the recumbent or Trendelenburg position. The sac or fundus of the diverticulum is situated above the neck and, therefore, unless the fundus is placed in a dependent position in relation to the neck, the diverticulum might not be opacified. If pulsion diverticula are not completely filled by food or liquid and if the neck of the diverticulum is not occluded, they will be easily visualized. Contraction of the diverticulum is indicative of the presence of muscular fibers in the wall of the diverticulum. The traction diverticula are more apt to show evidence of contraction.

Roentgen visualization of esophagobronchial fistulae can be accomplished by means of esophagography or bronchography. Bronchography with the aid of iodized oil is probably preferable to esophagography if barium sulfate mixture is used in the latter. The barium sulfate mixture might cause serious sequelae but no deleterious effects were noted in our case. The roentgen findings can be confirmed by esophagoscopy or bronchoscopy. These methods offer further opportunity to determine etiology by virtue of the facts that direct visualization is possible and that biopsy can be obtained.

TREATMENT

The cauterization method of therapy employed in our case was devised by Clerf who in 1933 reported a case of bronchoesophageal fistula treated with 30 per cent silver nitrate. This material was applied to the esophageal end of the fistula through an esophagoscope. The patient was then fed via a Rehfuss tube. Several weeks later, the orifice of the fistula appeared to have been obliterated. Clerf, Cooley and O'Keefe in 1943 reported 2 cases in which esophagoscopic application of sodium hydroxide crystal fused on a curved metal applicator was used with satisfactory results. Pape noted that his patient showed a decrease in complaints and that the fistulous tract was narrowed following such treatment. Whether the added factor of a diverticulum between the esophagus and air passages directly modifies the results obtained with Clerf's cauterization method as compared with the good results in simple bronchoesophageal fistulae is difficult to state. Beneficial effects have been noted following one cauterization but complete cures might require more than one application of caustic material in those cases where a diverticulum is present or in those instances where the fistulous tract is quite large.

The cauterization method, in our opinion, holds promise for cure of esophagobronchial fistulae. On the basis of our limited experience and review of the scant literature on the subject, it becomes evident that several factors must be considered and certain precautions taken. The application of the caustic must be carefully timed. A lengthy application is probably more harmful than a short one; too extensive or prolonged cauterization might result in erosion of a neighboring blood vessel. Following the cauterization it is important to put the esophagus at rest. This can be accomplished by inserting a stomach tube which should be left in situ for probably at least two weeks. Antispasmodics should be administered, and parenteral feedings are valuable adjuncts. Coughing should be prevented as much as possible by the administration of drugs such as codeine.

SUMMARY

A case of esophagobronchial fistula occurring at the site of an esophageal diverticulum is presented. The treatment of such

lesions according to the cauterization method of Clerf is discussed and the result of single cauterization of an esophagobronchial fistula through an esophageal diverticulum is described.

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TRUE PERICARDIAL DIVERTICULUM*

REPORT OF A CASE, WITH SAFE OPERATIVE REMOVAL

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HISTORY AND PATHOLOGY

PERICARDIAL diverticulum was first described in 1837 by Hart,[1] in the *Dublin Journal of Medical Science*. One hundred years later, Cushing,[2] in a comprehensive review, listed a total of 40 cases. Thirteen other cases have appeared in the literature since 1937, a total of 53.

In recent years, a very sharp and clearcut distinction has been made between two groups of these cases. First, there is the larger group of encapsulated pericardial effusions, bearing the same relationship to the pericardium as a loculated empyema does to the pleura. These "inflammatory diverticula" or "pseudodiverticula" occur in the pathogenesis of tuberculous pericarditis, rheumatic pancarditis and syphilitic cardiovascular disease. The second group, the so-called "congenital diverticula," occur in the absence of any other cardiac or pericardiac pathologic condition. Haas states that formation of such a diverticulum occurs at a point of weakness in the parietal pericardium, where the fibrous layer goes out along the great vessels. He describes three points of predilection; the base of the aorta high on the right side, the junction of the superior and inferior venae cavae on the right, lower down, and on the left, at the junction of the pulmonary veins. This group might well be labeled "true pericardial diverticula," so that their radical difference from encapsulated pericardial effusion in respect to pathogensis, appearance, and especially prognosis may be firmly established.

True diverticula are apparently the less common type; of the total of 53 cases reported, 38 (72 per cent) are encapsulated pericardial effusions; several case reports in the remaining group do not give sufficient information to classify them. No case of true pericardial diverticulum, proved by operation or autopsy, has appeared heretofore in the American literature.

CASE REPORT

The patient, female, aged fifty-five (Unit No. 340540) was first admitted to the Massachusetts General Hospital on February 2, 1942, for study. The patient was unmarried, American born, and a professional proofreader for many years. Her chief complaints were long-standing unproductive cough, and occasional epigastric distress, accompanied by mild nausea. There is no record of the presence of chest pain, dyspnea, cyanosis, expectoration, ankle edema, or weight loss. The past medical history included a hysterectomy fifteen years before. There was no history of tuberculosis, rheumatism, venereal disease, or glandular enlargement. Physical examination revealed mild obesity, blood pressure of 140/95, edentia, and normal body temperature, pulse, and respiratory rate. Hemoglobin was 15.5 gm. per 100 cc., erythrocyte count, 5,010,000, leukocyte count, 8,800, with 75 per cent polymorphonuclears, 21 per cent lymphocytes, 3 per cent monocytes, and 1 per cent eosinophils. On routine blood smear, the red blood cells and platelets appeared normal. Sedimentation rate was 5 mm. in sixty minutes. The blood sugar was 71 mg. and the nonprotein nitrogen 26 mg. The Hinton test was negative. A single urinalysis failed to reveal any abnormality. Kidney function, as expressed by the phenolsulfonphthalein test was good, 25 per cent of the dye being excreted at the end of fifteen minutes, and 55 per cent at the end of sixty minutes. The basal metabolic rate was minus 5 per cent. Stool examination was negative for blood and mucus. Oral cholecystography, barium meal, and barium enema failed to show abnormality in the gastrointestinal tract.

Examination of the chest (Fig. 1) revealed a sharply defined, pear-shaped mass, 6 cm. in its greatest diameter, lying in the right cardio-

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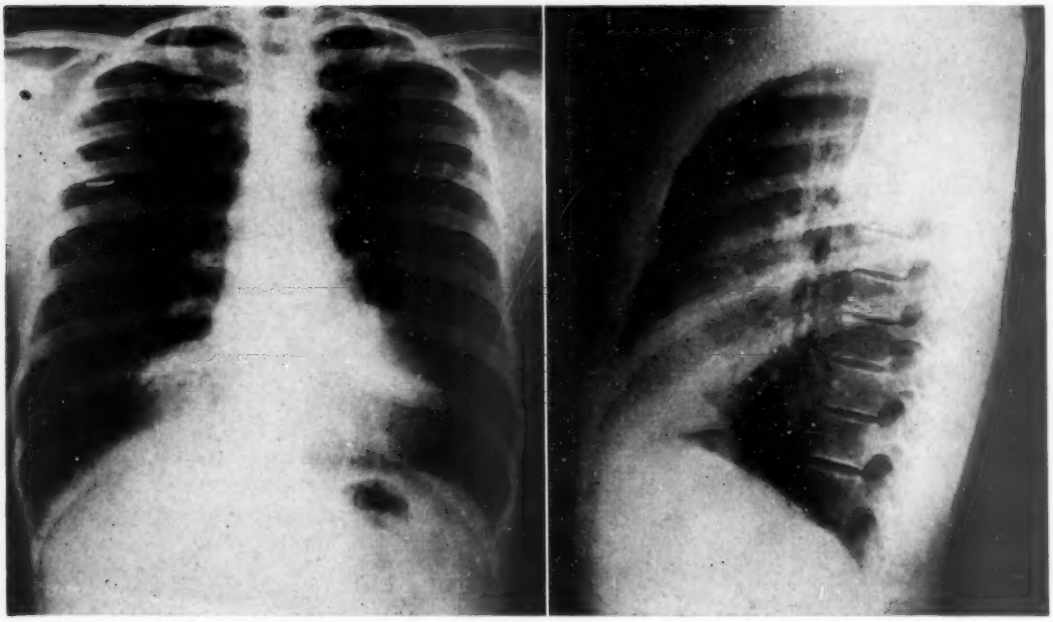

Fig. 1

phrenic angle, against the anterior chest wall. Under the roentgenoscope, the mass moved with the diaphragm; it did not pulsate, nor change size with respiration or change in position. The differential diagnoses considered were fat pad, omental hernia, and mediastinal tumor.

The patient left the hospital untreated, and was observed at intervals by her local physician. She was readmitted to the hospital eleven months later, on December 29, 1942. There had been no increase in the symptoms, and no new ones. Physical examination revealed no change from the findings previously recorded. Roentgenographic examination of the chest showed the mass to be slightly larger than before. On January 2, 1943, the chest was explored.

Operative Findings (Dr. Richard Sweet, surgeon). The chest was opened by an anterior intercostal incision. The tumor was easily found and was obviously cystic in nature. There was no thickening of the wall and no evidence of inflammatory reaction. The only attachment of the mass was to the pericardium. In developing the cleavage plane between the mass and the pericardium, it was noticed that the fluid within the mass began to disappear, and, in squeezing it, all the fluid was expressed into the pericardial cavity. When the pressure was released, the action of the heart slowly pushed the fluid back into the sac, distending it once more about as much as when it was first seen. This maneuver was repeated several times. The diverticulum was then thoroughly freed, and its communication with the pericardium found. The communicating opening was a little more than 0.5 cm. in diameter. At this point it looked very much like a hernial sac, and the coarse fibers of the pericardium could be seen decussating around the neck of the diverticulum, the wall of which was much thinner than the pericardium. There was no evidence of other disease of the pericardium. The neck of the sac was then opened and the communicating opening actually visualized. The diverticulum was removed and the chest wall closed. The patient had an uneventful convalescence, and was discharged from the hospital on the fourteenth postoperative day.

Pathological Findings. Grossly, the specimen is a roughly retort-shaped sac, measuring 8 by 4 by 1.5 cm. Its external surface is smooth, glistening, thin, and translucent. There was 22 cc. of light yellow, slightly blood-stained fluid in the sac. Microscopically, the lining is composed of well differentiated serosal cells.

DIFFERENTIAL DIAGNOSIS

True pericardial diverticulum is included in the group of diseases presenting themselves as a mass seen in, on, or close to the

heart. These include marked enlargement of one or more chambers of the heart, especially of the left atrium; aneurysm of the heart; tumor of the heart, pericardium, mediastinum, or adjacent lung, especially mediastinal dermoid or benign pericardial adenoma; encapsulated pericardial effusion; aneurysm of the root of the aorta; diaphragmatic hernia close to the heart, and fat pad.

The absence of clinical findings of cardiovascular disease will rule out marked enlargement of one or more of the chambers of the heart, cardiac aneurysm and encapsulated pericardial effusion. Aneurysm of the root of the aorta may be ruled out by absence of other evidence of cardiovascular syphilis, negative serology, and the roentgenological appearance of the rest of the aorta. Barium studies of the gastrointestinal tract will sometimes eliminate the presence of diaphragmatic hernia, depending on what is in the hernial sac. The history, the absence of physical signs in the chest, and possibly lipiodol bronchography will rule out lung tumor.

Differentiation from cardiac, pericardiac, or mediastinal tumor, especially dermoid or benign pericardial adenoma, and from fat pad appears more difficult. Location of the lesion is not especially helpful. The theory of Haas[3] as to the sites of predilection, mentioned previously, is confirmed by the fact that the majority of pericardial diverticula occur at the lower right border of the heart, anteriorly, a smaller number higher up on the right border and a smaller number near the location of the left atrium on the left.

Jansson[4] has found a very helpful sign: in his case, there was marked change in the shape of the mass in deep inspiration and forced expiration. In inspiration, the mass became long and narrow; in expiration, rounder, shorter, and broader. No such change was observed in our case; however, the diverticulum described by Jansson was four to five times larger than the one described here.

Dr. George W. Holmes[5] has suggested an interesting possibility in the differential diagnosis; that is, observation in the lateral decubitus position, with the mass higher than the heart, may show diminution in the size of the mass, since the fluid may run back into the pericardial sac, as observed at the time of operation in this case.

SUMMARY

A distinction has been offered between inflammatory sacculation of the pericardium, occurring in the pathogenesis of pericarditis, and true non-inflammatory diverticulum of the pericardium. A case of the latter type is reported.

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RICKETS AND INFANTILE SCURVY OCCURRING IN A CASE OF OSTEOGENESIS IMPERFECTA*

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AFFECTIONS of the skeleton may occur simultaneously in children. Examples of this are the combination of rickets and infantile scurvy, congenital syphilis and infantile scurvy, and rickets and lead poisoning.[1] Of these, the occurrence of rickets and scurvy is not infrequent, the development of rickets in a case of congenital syphilis is probably less frequent and the combination of lead poisoning and rickets is rare. A patient was admitted to the Children's Hospital who presented evidence of osteogenesis imperfecta with pathological fractures. At the age of three months he developed rickets and at the age of one year and nine months he was admitted with unmistakable evidence of infantile scurvy.

CASE REPORT†

The patient was a Negro male infant, aged three months, admitted for the first time on February 19, 1943. The baby was born at full term with an easy labor and a spontaneous delivery. His birth weight was 6 pounds. At two months of age his mother noticed that his head was soft. He had been breast fed for one month and then was given evaporated milk, 8 oz. with two tablespoonsful of karo and water, 13 oz. The mother claimed that orange juice was started at two months and had been given faithfully. He also received, starting at two months, ½ teaspoonful of cod liver oil, daily.

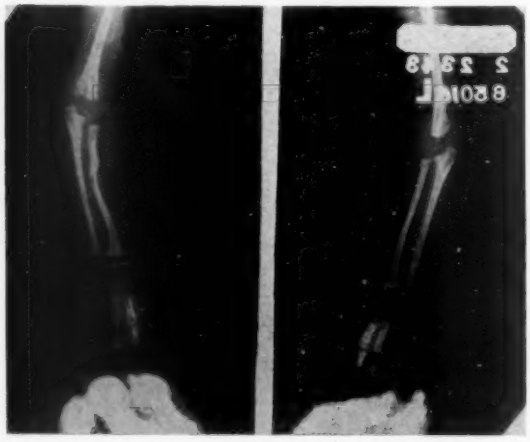

FIG. 1. Note the deformity of the right radius with decalcification and an osteoid zone at the distal end of the diaphysis. Slight flaring of the distal extremity, right radius. Examination made on first admission of patient to the hospital.

The infant was underdeveloped and undernourished but did not appear to be acutely ill. The head had a soft consistency suggesting a craniotabes. The circumference of the head was $13\frac{3}{4}$ inches, the anterior fontanelle measured $1\frac{1}{4}$ by $\frac{1}{4}$ inches, the posterior fontanelle $\frac{1}{2}$ by $\frac{1}{2}$ inch. The fontanelles were not bulging. On this first admission no bluish tinge of the scleras was noted. In the chest there was a slight prominence of the costochondral junctions. The heart and lungs were normal.

The Wassermann and Kahn reactions were negative. On admission, the serum calcium was 9.2 mg., serum phosphorus was 3.8 mg., phosphatase 17.9 Bodansky units. The blood urea nitrogen was 11.5 mg. By April 23, 1945, the serum phosphatase had dropped to 14.8 Bodansky units.

On admission, the roentgen examination revealed thinning of the cortex of all the long bones. In the forearms decalcification of each radius and ulna was shown, the bones having a slightly streaked appearance. The right radius was curved and deformed which could have been due to old fracture, possibly intrauterine. The distal ends of both bones of each forearm showed spreading, especially the right. The distal end of the diaphysis of the right radius was cupped with a layer of osteoid bone at its extremity, and there was fraying out of the zone of temporary calcification.

In the thorax there was marked atrophy and thinning of the ribs with spreading and cupping of their anterior ends. A fracture of the right clavicle was present with callus formation. Each humerus also was atrophied.

† I wish to express my thanks and appreciation to Dr. Joseph Stokes for permission to report this case from his service.

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In the skull the tables were extremely thin with an open anterior fontanelle and somewhat widened sutures.

At the time of discharge, after treatment with high dosage of vitamin D and calcium chloride, the infant had gained weight and was much improved. The roentgen examination showed an improvement in the degree of calcification of the bones of the forearms, increased density of the zones of temporary calcification and a disappearance of the osteoid zone at the

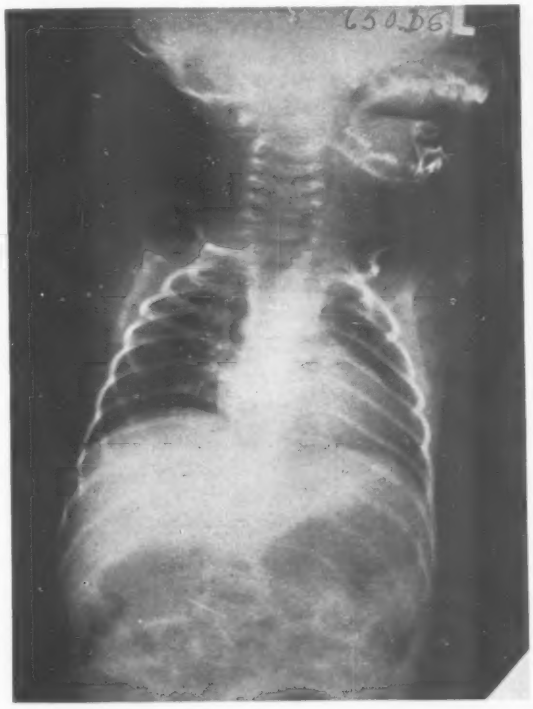

Fig. 2. Marked thinning and atrophy of the ribs with flaring of their anterior ends. Old fracture of the right clavicle. First admission.

distal end of the diaphysis of the right radius. The patient was discharged on April 30, 1943.

The patient was admitted the second time on August 28, 1944, with a history of illness since August 1, 1944. He was extremely fretful and cried when handled. He lay with both legs drawn up in a frog position. For three days before admission his mother had noticed blood in the stools. She gave a history of adequate feeding except that the child took no orange juice. The patient's weight was 14 pounds. On physical examination he appeared to be acutely ill, was malnourished and underdeveloped. The head gave the impression of possible cranio-

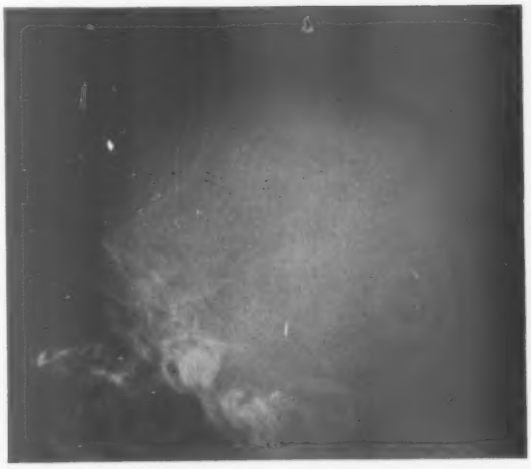

Fig. 3. Marked thinning of all bones of the vault of the skull with lack of normal calcification persisting after seven weeks of antirachitic therapy.

tabes. A slight bluish tinge was noted in the scleras. The gums were sore and bleeding. In the chest there were enlarged costochondral junctions but no Harrison's groove. All joints were tender and enlarged.

On August 29, serum calcium was 9.4 mg. and phosphorus was 4.1 mg., phosphatase was 6.8 Bodansky units.

On roentgen examination the long bones showed extreme thinning of the cortex, and atrophy more marked than on the first examination. The atrophy was especially prominent in each fibula. Typical scorbutic hemorrhages

Fig. 4. Increased calcification of the diaphyses of the bones of the forearms with disappearance of the osteoid zone at the distal end of the right radius, after seven weeks of antirachitic therapy.

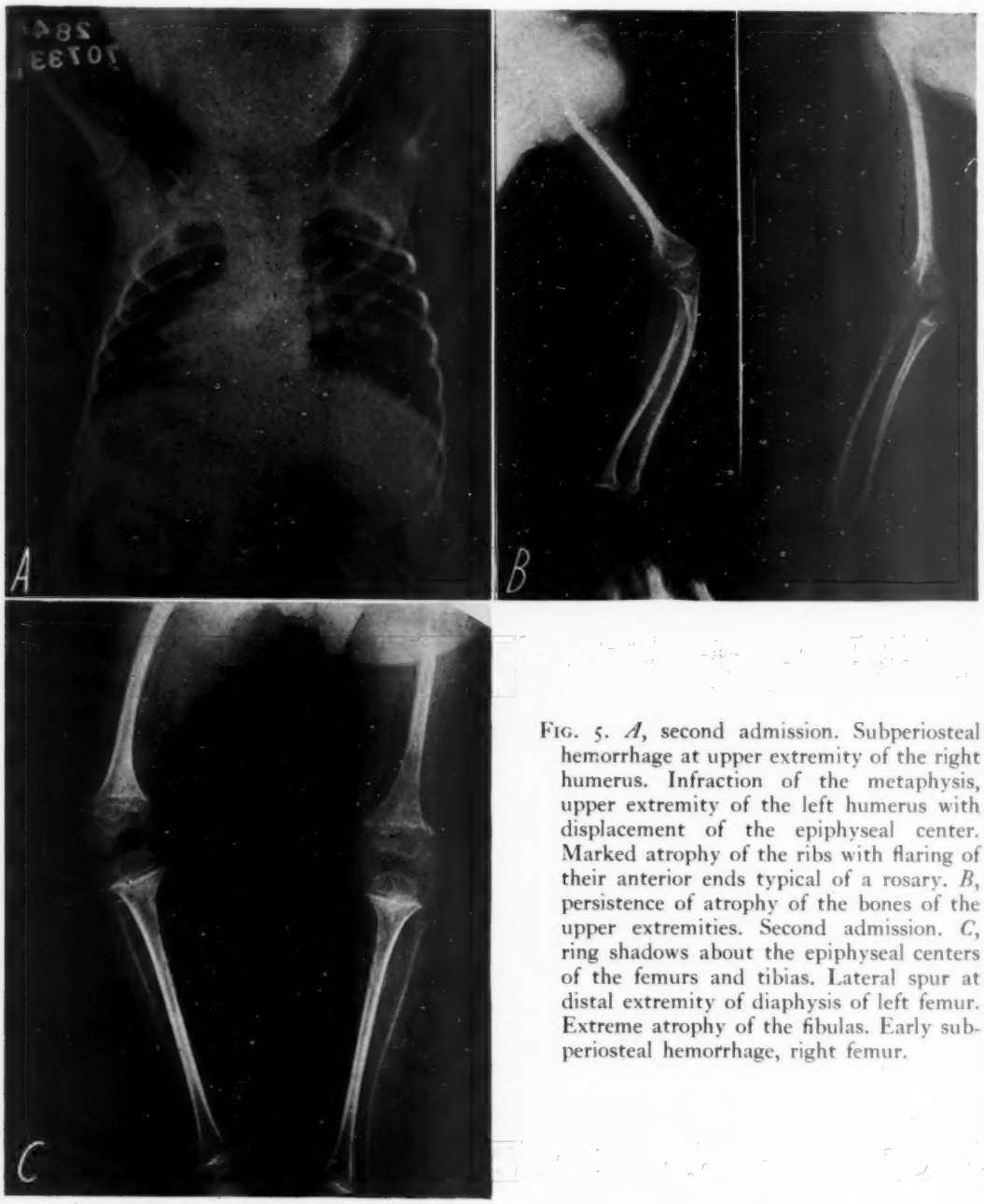

FIG. 5. *A*, second admission. Subperiosteal hemorrhage at upper extremity of the right humerus. Infraction of the metaphysis, upper extremity of the left humerus with displacement of the epiphyseal center. Marked atrophy of the ribs with flaring of their anterior ends typical of a rosary. *B*, persistence of atrophy of the bones of the upper extremities. Second admission. *C*, ring shadows about the epiphyseal centers of the femurs and tibias. Lateral spur at distal extremity of diaphysis of left femur. Extreme atrophy of the fibulas. Early subperiosteal hemorrhage, right femur.

were present in the upper end of the diaphysis of the right humerus and in the distal end of the diaphysis of each femur. In the upper end of the diaphysis of the left humerus there was a metaphyseal infraction with displacement of the epiphyseal center. Distinct ring shadows were visible in the epiphyseal centers of the upper ends of the tibias and the lower ends of the femurs.

In the thorax marked spreading and cupping of the anterior rib ends with extreme thinning and atrophy were present. The appearance was that of a rosary, most likely of scorbutic origin. The old fracture of the right clavicle was still visible.

The tables of the skull were better calcified than on the first admission but they were still somewhat thinner than normal. A mosaic ap-

Fig. 6. *A*, subperiosteal hemorrhages in the distal ends of the diaphyses of the femurs, one month after antiscorbutic treatment was started. *B*, appearance of the skull, one month after second admission (age, twenty-two months). Note thinning of the tables with mosaic appearance of the occipital area.

pearance, due to large wormian bones in the occipital and posterior parietal areas had developed since the last examination. The sutures were again wider than normal.

The child was placed on a high caloric diet and was given therapeutic doses of all vitamins. On discharge his legs were fairly well extended and roentgen examination showed satisfactory healing of the scorbutic hemorrhages. His gain in weight was not, however, satisfactory and he had only gained 1 pound when discharged on November 11, 1944.

He was admitted for the third time on November 11, 1944. He had fallen from a highchair the day previous to admission. He held his left leg immobile. His mother stated that she had put him to bed immediately after the fall where he remained without complaint. The roentgen examination showed a fracture of the left femur in the distal third with some bowing of the fragments, convexity anterior. The rings about the epiphyseal centers of the femur and tibia had become denser and wider and there was increased calcium deposition in the cortex of the shafts. Atrophy was still present, especially in the fibula. He was discharged on December 13, 1944. The bluish tinge of the scleras was again noted on this admission.

On February 4, 1945, he was admitted for the fourth time. There was no history of a definite trauma. On roentgen examination a fracture of the right humerus was found located at approximately the junction of the upper and middle thirds of the bone. He had been in good con-

Fig. 7. Well developed subperiosteal hemorrhage in healing stage, upper end of the right humerus after six weeks of antiscorbutic treatment during second admission. Note healing of the infraction in upper metaphysis of the left humerus.

FIG. 8. Third admission. Fracture of the left femur in the distal third. Increased density and width of the rings about the epiphyseal centers and of the zones of temporary calcification.

dition from the date of his previous discharge until three days before admission when he developed a severe cough and had refused to eat. His mother brought him to the hospital because of this and not because of any symptoms referable to the fracture. On both the third and fourth admissions he had a chronic otitis media. He was discharged on March 14, 1945.

DISCUSSION

Osteogenesis imperfecta tarda is characacterized roentgenologically by the typical gnarled appearance of the bones of the extremities with marked deformities caused by the multiple fractures. The cortex is thin. Callus deposits can be detected in the shafts at the sites of the fractures. The thorax is compressed and the ribs are bent. The bones usually show a marked degree of osteoporosis. The tables of the skull are extremely thin and often calcification and ossification seem to be entirely lacking in some areas. Sutures are wide.

Clinically the skull on pressure is no thicker than parchment due to defective membranous bone development. This has led to the term "rubber ball head." Knaggs[6] called attention to the fact that the membranous areas which intervene between the immature bone are apt to become filled with numerous wormian bones resulting from discrete patches of ossification. The vault is thus represented by a mosaic of larger and smaller bone plates, sometimes touching one another and sometimes united by bridges of periosteum and dura. In some cases, the skull can be moulded by pressure of the hands.

Osteogenesis imperfecta tarda is characterized in the roentgenogram by marked thinning of the cortex of the long bones and deformity usually dependent upon the number of fractures present. There may, however, be bowing without actual fracture. Generalized osteoporosis may be seen. The metaphyses and epiphyses have a

FIG. 9. Fourth admission. Fracture of the right humerus. Persistence of atrophy of shafts.

widened appearance for the most part due to the atrophy of the shafts. The area of the diaphyso-epiphyseal junction is usually normal. Slight infractions of the periosteum may be seen sometimes rather than actual fracture. In such areas callus formation can later be found. There is often a noticeable lack of pain accompanying the fractures. This has been accounted for by the subperiosteal nature of many of the fractures. Knaggs believes that, in the cases in which signs first appear in childhood or adolescence, the fundamental change was present in early uterine life. The less the defect, the greater is the likelihood that its signs will be delayed until the patient is able to get about and is exposed to the ordinary slight traumatisms of a usually healthy life. As a rule, the earlier in life the fractures first occur, the greater is the liability to fracture.

The diagnosis of osteogenesis imperfecta can be justified by the following: the occurrence of multiple fractures, some without history of trauma; the roentgen appearance of extreme atrophy of the shafts of the long bones and ribs and the apparent widening of the epiphyseal and diaphyseal areas; the lack of pain accompanying the fractures; the bluish tinge of the scleras. No history of occurrence in other members of the family could be obtained but this absence of a familial or hereditary tendency does not rule out the diagnosis as the nonhereditary occurrence of osteogenesis imperfecta or osteopsathyrosis has been reported quite frequently in the literature. Such cases have been included in a separate category in the classification of the disease by Key.[5]

The presence of the fracture of the right clavicle and the deformity of the right radius suggests that the condition was present at birth and during intrauterine life. The roentgen appearance is not that of the usual type of osteogenesis imperfecta congenita but is more like that of the late types. The unusual feature is the presence of this roentgen appearance in early infancy with some evidence that it was present at birth. Foote[3] has described an infantile type that appears after the child begins to walk or stand without support. These children usually die comparatively early as a result of intercurrent infection.

The signs indicative of rickets on the first admission of the patient are as follows: decalcification of the bones of the forearms and lower extremities; the fraying out of the zones of temporary calcification in the distal end of the right radius with the presence of an osteoid zone; the tendency to widening and spreading of the diaphyseal ends and the similar appearance in the rib ends due to enlargement of the costochondral junctions.

The craniotabes of the skull could be due either to rickets or to osteogenesis imperfecta. Park and Eliot[7] state that the craniotabes of osteogenesis imperfecta is fully developed at birth, is far more extensive than in rickets and involves the anterior half of the skull as well as the posterior half. In osteogenesis imperfecta there are sometimes scattered islands of bone. They also call attention to the fact that in normal infants, one or two months old, but more frequently in premature babies, thinness of the skull may be found which is of no pathological significance. In the skull of the case reported here there was only a uniform thinning of the tables with lack of calcification. On the second admission a mosaic appearance had developed which suggests involvement due to osteogenesis imperfecta. It would seem logical to assume that the rickets was of mild degree and that the condition of the skull was not due solely to rickets. However, increased calcification and slight thickening of the tables occurred after antirachitic treatment was begun.

The occurrence of subperiosteal hemorrhages confirms the diagnosis of infantile scurvy. In addition, there were definite ring shadows about the epiphyseal centers. Other signs such as thinning of the cortex, the ground glass appearance of the metaphyseal areas extending toward the middle of the shafts and the zone of rarefaction adjacent to the zone of temporary calcification, were obscured by the markedly

atrophic condition of the bones present from birth. The spreading and cupping of the rib ends due to marked enlargement of the costochondral junction found on the second admission could have been due to a scorbutic rosary.

Czerny and Keller[2] drew attention to multiple fractures with general fragility of the bones occurring in infants which they ascribed to a special form of avitaminosis and which they believed should not be included in the category of osteogenesis imperfecta of either the congenital or late types. This was later emphasized by Heise[4] who suggested that as blue sclera is an indication of originally weakened mesenchyme, the patients are less resistant to bone diseases in general. He reported the cases of two children, twins, who after attacks of scurvy showed fragility of the bones with fractures. He felt that they could not be considered as cases of fragility due to osteogenesis imperfecta but of fragility due to avitaminosis. The fact that fractures were present before the occurrence of the scurvy would rule out such cause in the patient under consideration.

SUMMARY

A case is reported of a male Negro infant who showed evidence of osteogenesis imperfecta apparently present from birth. Rickets developed at the age of three months and infantile scurvy at twenty-one months. The unusual feature of the case is the presence at an early age of osteogenesis imperfecta with some evidence that it was present from birth and that the type of roentgen changes in the skeleton was indicative of the late type of the disease rather than the congenital.

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GENERALIZED LEONTIASIS OSSEA*

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LEONTIASIS OSSEA is an uncommon disease, consisting of diffuse bony thickening, usually confined to the skull (notably the bones of the face) but occasionally associated with involvement of the long bones. It is also known as hyperostosis of the skull, megalocephaly, fibromatosis osteoplastica ossium and hyperostose des os de la tête. The condition was first described by Malpighi in 1697 but was not called leontiasis ossea until 1864 when Virchow described it in considerable detail. It is said that the name was chosen because the bony overgrowth corresponded with the overgrowth of connective tissue seen in molluscum fibrosum with leonine facies.[3] The hyperostosis may be limited to one bone of the skull or involve all of them; in the latter event, the bones of the face may be much enlarged and distorted, but the facies, despite the name, is almost *never* leonine. Skulls of considerable thickness (calvarium of 5 cm.), and weight (4,000 gm.) have been reported.

The disease begins in childhood or adolescence. Females are said to be more commonly afflicted than males.

The cause of the disorder is unknown. Infectious, metabolic and endocrine origins have been postulated but none proved. Because some cases, both grossly and microscopically, have resembled cases of osteodystrophia fibrosa hyperostotica (Paget's disease), the condition has been erroneously regarded as a cranial form of osteitis deformans.

PATHOLOGY

The process may involve one or all the bones of the skull, and sometimes, the long bones. Grossly, Knaggs[6] divided it into two types, the creeping periostitic and the diffuse osteitic type. The histopathological changes are the same in both, but subperiosteal bony deposits are larger in the periosteal type. The following pathological description is given by Bauer:[1]

Early in the disease one notes disappearance of the usual bony structures. The outer layers are less compact, and areas of bone dissolution appear filled with vascular and fibrous connective tissue which may subsequently become fibrosed. New bone may be laid down in irregular fashion in such fibrous areas, although neither the laminations nor the haversian systems of normal bone are formed. Small cysts may remain. In the later stages, the bone loses its normal contour and is greatly thickened and porous. When suppuration is present, it is the result of secondary infection. Isolated bone sections may resemble either Paget's disease or osteitis fibrosa cystica except that the fibrosis is more marked in leontiasis ossea.

SYMPTOMS

The symptoms vary widely, for no obvious reason. Some cases, with slight involvement of the skull, allege that they suffer marked headache and various visual or auditory disturbances; others, with extensive hyperostosis, are virtually asymptomatic. The overgrowth of the malar or mandibular bones produces facial deformity which may disturb the patient more or less in proportion to his amour propre.

Headache is sometimes a severe symptom. Exophthalmos is frequently present. Visual and hearing defects, neuralgia, and nasal and lacrimal duct obstruction have been reported. Insomnia, mental dullness and convulsions may develop. The course of the disease is quite variable (from twenty to forty years). Death has been reported from cachexia and from convulsions.[3]

DIAGNOSIS

The diagnosis is usually made by roentgen examination of the skull, with the finding of diffuse hyperostosis of the bones of the face (notably the malar, frontal and

* The opinions and views set forth in this article are those of the writer and are not to be considered as reflecting those of the Navy Department.

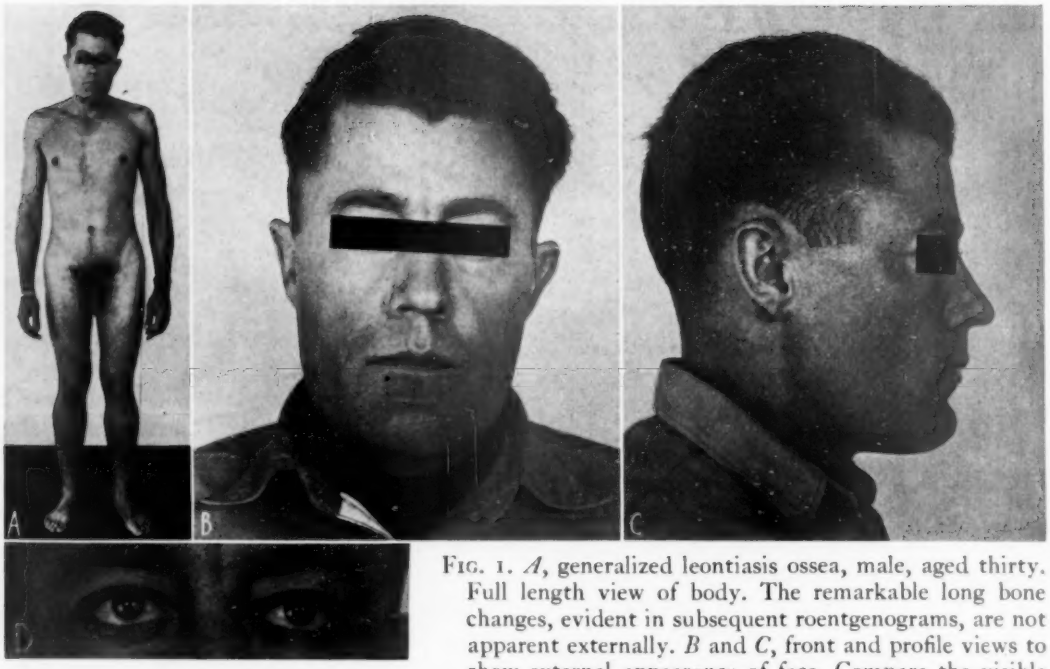

FIG. 1. *A*, generalized leontiasis ossea, male, aged thirty. Full length view of body. The remarkable long bone changes, evident in subsequent roentgenograms, are not apparent externally. *B* and *C*, front and profile views to show external appearance of face. Compare the visible size of the mandible with that seen in the subsequent roentgenograms. *D*, close up of eyes. The degree of exophthalmos was more apparent than the photograph shows.

sphenoidal bones). There are variable degrees of actual bony overgrowth and occasionally enormous thickening of these bones. The hyperostosis is usually uniform and "osteomatous," and not associated with macroscopic areas of porosis or cystic change. The mandible and any of the other bones of the skull may be involved. Rarely, there is diffuse cortical hyperostosis of the long bones of the extremities, usually bilateral and symmetrical in distribution.

The differential diagnosis includes chronic inflammatory osteitis (pyogenic or syphilitic), Paget's disease and acromegaly. We doubt if chronic radium poisoning or chronic fluorosis would require differentiation. In inflammatory osteitis the involvement is usually not bilateral and symmetrical (as it tends to be in leontiasis ossea) and sequestra may be present. In Paget's disease, the characteristic nummular areas of porosis and sclerosis, plus the diffuse striated nature of the hyperostosis, will usually serve to distinguish the entities. Further, in the latter, the pelvis, spine or long bones will frequently show characteristic changes that help to identify the process. Leontiasis ossea tends to appear in younger age groups, Paget's disease in older persons. In acromegaly the jaw bone is mainly elongated and hypertrophied, not grossly hyperostotic or osteomatous; the sella turcica may be enlarged; the hands will show characteristic growth changes, both soft tissue and phalangeal. The entire skeleton may be increased in size.

Inasmuch as leontiasis ossea with involvement of the long bones is a rare clinical entity, it is deemed worth while to place the following case on record.

CASE REPORT

The patient was a white male, aged thirty, a machinist's mate in a Construction Battalion. He first sought medical attention on account of intermittent frontal headaches and progressive enlargement of the jaw.

Present Illness. At first, he stated that his headaches and jaw changes were only of one year's duration (he had enlisted about fourteen months prior to his visit to sick bay); however, on further questioning, he admitted

Fig. 2. Generalized leontiasis ossea. *A*, frontal; *B*, dorsal; *C*, lateral, and *D*, Waters projection of the skull. Note the hyperostotic changes in the frontal, sphenoidal, and temporal bones; the petrous apices show dense sclerosis; the upper portions of the parietal bones and the central portion of the occipital bone show moderate hyperostosis.

he had had headaches for a period of about five years, especially on stooping over, and that his jaw had begun enlarging about two and one-half years ago. His eyes had been more prominent than usual for a similar period, and the hearing in his left ear had been getting

slowly worse during that time. He had concealed these symptoms at time of enlistment, being then anxious to escape from some domestic difficulties. His bilateral exophthalmos had been noted at time of induction, but not his prognathism.

Previous History. Aside from that noted under present illness, this was not remarkable. He had no childhood diseases or injuries of note; no history of lues. He was born in Nebraska. He had worked as a farm hand in his youth and in construction jobs since that time.

Family History. His father and mother died when he was an infant, of causes unknown. Both were natives of Nebraska; neither had enlargement of the jaws or other bones, as far as he was aware. He has one brother, aged thirty-nine, with "such extensive enlargement of the jaw that he was rejected for military service"; this brother was told "by an Omaha clinic that he had an incurable bone disease, and is being treated for acromegaly." The patient is married and has two children, living and well, without bony deformities.

Physical Examination. A well developed male, with a slightly prominent jaw, in no apparent distress (Fig. 1, *A*, *B*, *C*). Height 5 feet, 10 inches; weight 160 pounds. Exophthalmos, bilateral, of a fairly marked degree, but without lid lag (Fig. 1*D*). Vision in both eyes 20/15. Ocular fundi negative. Visual fields and acuity normal. Ears externally negative; audiograms revealed slight impairment of hearing in left ear; right ear normal. Marked symmetrical enlargement of the mandible, not quite so pronounced at the symphysis as elsewhere. No tenderness, nor limitation of motion of the jaw. Remaining bones of face, grossly negative. Reflexes negative. Temperature 98.6°F., pulse 88, respiration 22, blood pressure 120/70.

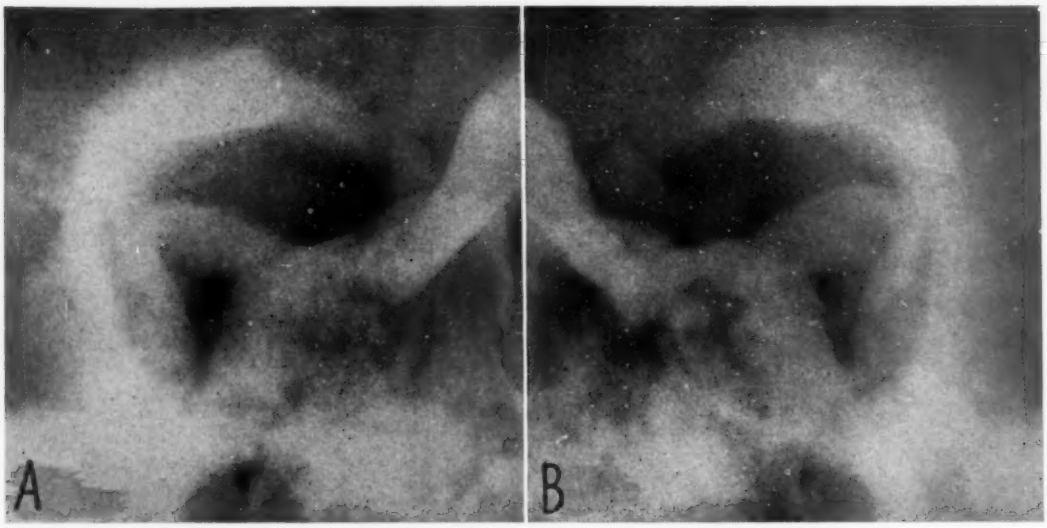

FIG. 3, *A* and *B*. Optic foramina. The optic foramina are slightly smaller than average (direct measurement on 36 inch target film distance roentgenograms being: left, 3.5×5 mm.; right, 3.5×4 mm.).

Laboratory Examinations. Red blood cells 4,140,000 per cu. mm. White blood cells 9,200 (65 per cent neutrophiles; 36 per cent lymphocytes). Sedimentation rate normal. Hemoglobin 14 gm. Blood calcium 10 mg. per 100 cc. Blood phosphatase studies not available. Kahn reaction negative. Urinalysis negative.

Roentgen Examination. Skull: There is diffuse and remarkable hyperostosis of the inferior half of the frontal bone, involving notably the orbital ridges and the floor of the anterior fossa; similar changes are present in the sphenoidal bone, the mandible, and portions of the malar and temporal bones (Fig. 2, *A*, *B*, *C*, *D*). There is slight generalized hyperostosis of por-

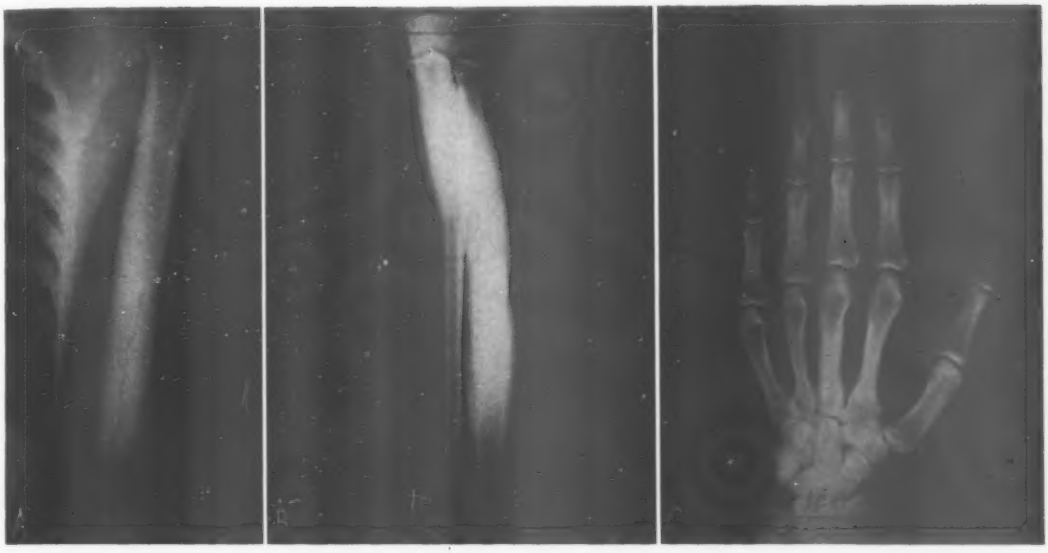

FIG. 4. *A*, left humerus; *B*, radius and ulna; *C*, wrist and hand. Similar changes were present in the right arm and hand. Note the diffuse thickening of the cortex of the humerus. The changes in radius and ulna resemble extreme osteoperiostitis. The hyperostosis of the shaft of the right second metacarpal was more marked than that of the left; otherwise the changes in the hands were identical.

tions of the remainder of the calvarium, especially at the vertex and about the external occipital protuberance. The diploe is virtually obliterated. The optic foramina appear to be a trifle narrowed (direct measurements on 36 inch target film distance roentgenograms being left 3.5×5 mm. and right 3.5×4 mm.) (Fig. 3, *A* and *B*). The auditory canals are not visibly narrowed, but portions of the temporal bones show the above mentioned hyperostosis.

Spine, Ribs, Sternum and *Pelvis:* Roentgen appearance normal (Fig. 5).

Chest: Heart vessel shadow negative; transverse cardiac diameter 12 cm. Healed primary complex in middle third of left lung. Lungs otherwise clear.

Kidney, Ureter, Bladder: Normal. No renal opacities.

Upper Extremities: Diffuse thickening of the cortex of each humerus, with virtual obliteration of the cancellous spaces in the distal half of each bone. The cortex reaches 23 mm. in thickness in certain areas. Diffuse hyperostosis, somewhat resembling the so-called "flowing" type, involving the proximal third of each ulna and the middle three-fourths of each radius. The appearance here is one of extreme osteoperiostitis. There is hyperostosis of the shafts of the second and third metacarpals of each hand (more marked on the right side) without evidence of involvement of the other bones of the hands at the present time (Fig. 4, *A, B, C*).

Lower Extremities: Diffuse thickening of cortex of the shaft of each femur, and of the shaft of each tibia and fibula. In places, notably on the tibiae, the appearance is one of "creeping periostitis." The cortex of each tibia shows major changes on its mesial, lateral and posterior aspect (not on its ventral) (Fig. 6, *A, B, C*). There is no patchy porosis or other

FIG. 5. The pelvic bones are not involved. Neither were the ribs, clavicles, sternum, nor vertebrae.

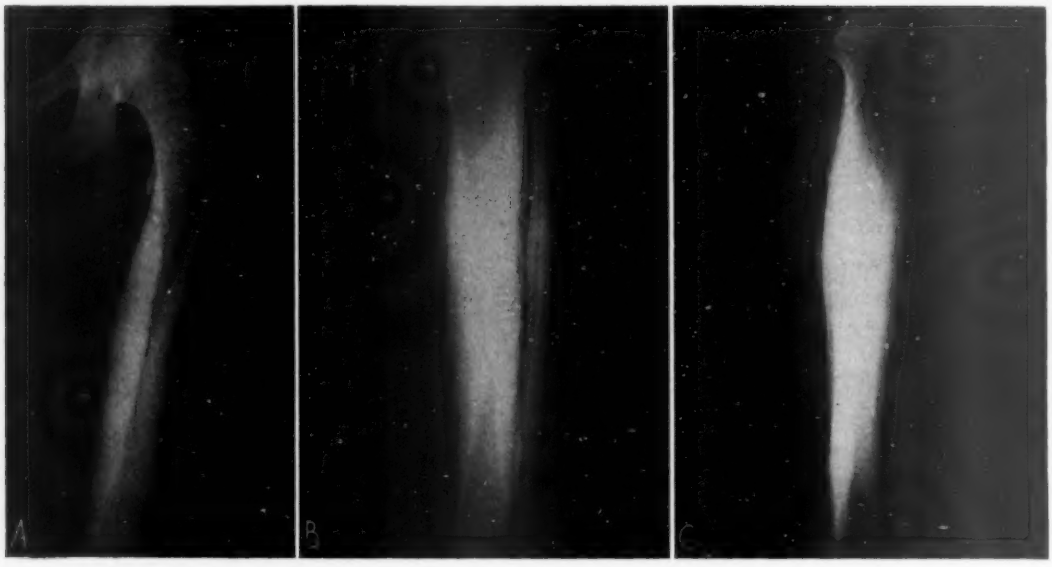

FIG. 6. *A*, left femur; *B*, tibia; and *C*, fibula. Similar changes were present in the right leg. The femoral thickening is essentially a diffuse cortical hyperostosis. The tibial changes are most marked on the dorsal, mesial and lateral aspects of the bone (in contradistinction to the usual location of the changes in Paget's disease). They are pseudo-inflammatory in appearance. The feet showed no bone changes.

evidence of Paget's fibrous dystrophy. Both feet are negative.

Conclusion: Generalized hyperostosis of portions of the skull and the bones of the extremities (generalized leontiasis ossea). The resemblance of the changes in the calvarium to those of Paget's disease and the slight resemblance of the changes in the arm bones to those of melorheostosis Léri is noteworthy, but we believe the basic lesion to be an unusual type of leontiasis ossea.

DISCUSSION

This case is of considerable interest for several reasons: (1) the virtual absence of involvement of the maxillae, (2) the extensive involvement of the bones of all four extremities and, especially (3) the fact that *both* of Knaggs' "types" of leontiasis ossea are present in one and the same patient, the osteitic (as shown best in the mandibular and femoral changes) and the periostitic (evident in the radial and tibial changes).

There is a surprising amount of confusion between Paget's disease, acromegaly and leontiasis ossea in the literature, even in authoritative textbooks.[2,4,5,7] In Ewing's "Neoplastic Diseases"[4] appears the sentence: "In acromegaly there is diffuse overgrowth of the bones of the skull and extremities, resulting from disturbance of hypophyseal function (leontiasis ossea)."

In Boyd's "Pathology"[2] we find the statement: "In Paget's disease the bones of the face are occasionally greatly thickened (leontiasis ossea)." MacCallum's "Textbook of Pathology"[7] carries an identical sentence.

In the introduction to "Roentgen Interpretation" by Holmes and Ruggles,[5] it is stated that in leontiasis ossea the mandible is *not* usually involved: "Leontiasis ossea or creeping periostitis is somewhat similar to hyperostosis frontalis. It may spread slowly over the maxillae, frontal and parietal regions. Usually, the mandible is not involved. The condition may be confused with Paget's disease."

We believe that the case under review is one of idiopathic (perhaps congenital) overgrowth of the cortical portions of many of the bones of the skull and extremities, and fits into the classification of generalized leontiasis ossea. Saucerotte[4]

is said to have reported such a case, but details thereof are not presently available to the writer. The type of hyperostosis, the absence of porotic changes and the anatomic distribution all favor the diagnosis given. If only a few of the involved areas of the skeleton had been examined, it might have been classified as the creeping periostitic type of Knaggs, while, if others were roentgenographed, it might have been classified as the diffuse osteitic type.

The prognosis of cases such as the one reported herewith is obviously poor. Should the exophthalmos be progressive, surgical decompression of the orbital cavities will presumably be necessary. Plastic procedures on the mandible are conceivable, though of dubious utility. Progressive hyperostosis of the long bones with obliteration of the marrow spaces will probably result in some degree of anemia. Continued changes in the temporal bones may result in further impairment of hearing.

SUMMARY

1. Leontiasis ossea is a rare but definite clinical entity, apparently not related to Paget's disease, acromegaly or other conditions sometimes associated with facial bone enlargement.

2. Leontiasis ossea is usually confined to the bones of the face and skull, but a small number of cases also show diffuse and remarkable cortical hyperostotic changes in the long bones of the extremities (generalized leontiasis ossea).

3. A case of generalized leontiasis ossea in a male aged thirty years is reported. The cranial bones and mandible showed typical diffuse "osteitic" overgrowth; the long bones of the extremities showed diffuse, symmetrical cortical and periosteal overgrowth.

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THE HISTOLOGIC EFFECTS OF RADIOPHOSPHORUS ON NORMAL AND LYMPHOMATOUS MICE*†

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IN EARLIER papers[11,12] the distribution of radiophosphorus (P^{32}) in normal and lymphomatous§ mice has been described. In addition, its effect on the normal blood forming tissues has been observed.[1,14] On the basis of these investigations P^{32} has been applied therapeutically in the treatment of chronic leukemia.[2,4,7,8,9,13,15] To gain further knowledge of the radiation effects of P^{32} we have used a transmissible lymphoma in mice.

PART I

Histologic and Hematologic Effects of P^{32}. Two series of experiments were performed on normal and lymphomatous mice as follows: In Series I, two groups of "A" strain mice (aged five months, sexes mixed) were used. In the first group of 13 mice, 7 received an intravenous injection of approximately 1.2×10^7 lymphoma cells.[10] The other 6 mice were used as controls. Of the 14 mice in the second group, 8 received a similar dose of lymphoma cells, and the 6 remaining mice acted as controls. (The age of this second group of mice was three months, and the sexes were mixed.)

Thirteen days after inoculation, 3 leukemic animals in each group were given 1.0 cc. of an isotonic solution of sodium phosphate intravenously which contained 195.3 microcuries‖ of P^{32} per c.c. Five control animals received a similar dose of P^{32} at the same time.

In Series II, 12 mice (three month old males) were given 1.2×10^7 lymphoma cells intravenously, and seven days later half of these animals received four 1.0 cc. subcutaneous injections (54.5 microcuries per cc.) at two hour intervals. Four normal animals received similar P^{32} injections and 2 additional mice were kept as controls.

Blood Studies. Cells with ring (doughnut) shaped nuclei were classified as "early granulocytes"; large lymphocytes with immature nuclei and abundant cytoplasm were classified as "early lymphocytes"; "large lymphocytes" includes monocytes; lymphoma or leukemic cells were identified by their large size, primitive nucleus, scarce and strongly basophilic cytoplasm. The white cell counts taken on the normal mice were subject to wide variation.

The course of the total white counts after P^{32} administration is shown in Figure 1. It will be noted that, in both series, the drop in white cell count in leukemic animals was greater than that in the normal during the first four days after injection with P^{32}. In the following five day interval (four to nine days after P^{32} administration) the drop of the white cells for Series I approached a plateau while the drop in Series II continued, but at a slightly diminished rate. Following this period the leukemic animals of Series I showed an increasing total count while those of Series II continued to decrease.

In Figure 2 is shown the differential effect of P^{32} on the white cells of normal mice. The small lymphocyte was at first the most markedly affected; while the mature granulocyte was comparatively little affected. Four days after P^{32} injection the drop of the lymphocytes reached a

§ When implanted subcutaneously, a local tumor develops followed by leukemic invasion. After intravenous injection of lymphoma cells, no local tumor develops but a leukemic picture is the result.[4]

‖ Beta-ray standard.

* This work was supported by the Columbia Fund for Medical Physics of the Columbia Foundation.

† This work was completed in 1940 but it was not possible to report it at that time, due to transference of our activities.

‡ Department of Physics.

plateau, while the granulocytes began to drop at a greater rate. This drop continued at least to the fourteenth day. However, after the ninth day the small lymphocytes showed a slight rise.

Figure 3 shows the effect of P^{32} on the white cells of leukemic mice. They were affected in a manner similar to that ob-

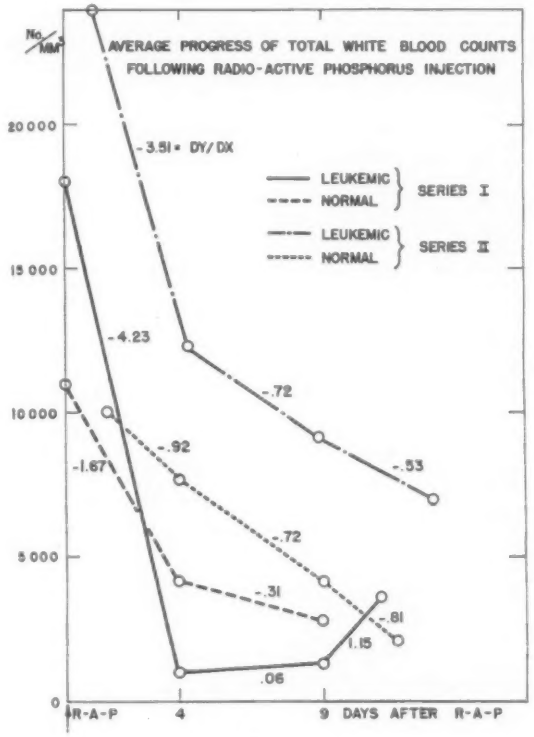

FIG. 1

served in the control animals. In addition, it can be seen that P^{32} was effective in decreasing the number of leukemic cells in the blood for as long as nine days after administration of this particular dose of P^{32}.

Red cell counts and hemoglobin determinations were not carried out, but with these relatively large doses of P^{32}, one would expect after a period of time very low red counts and hemoglobin values.[8] It should be pointed out that the lethal dose for mice is in the neighborhood of 70 microcuries. The doses used in these hematological and histological experiments

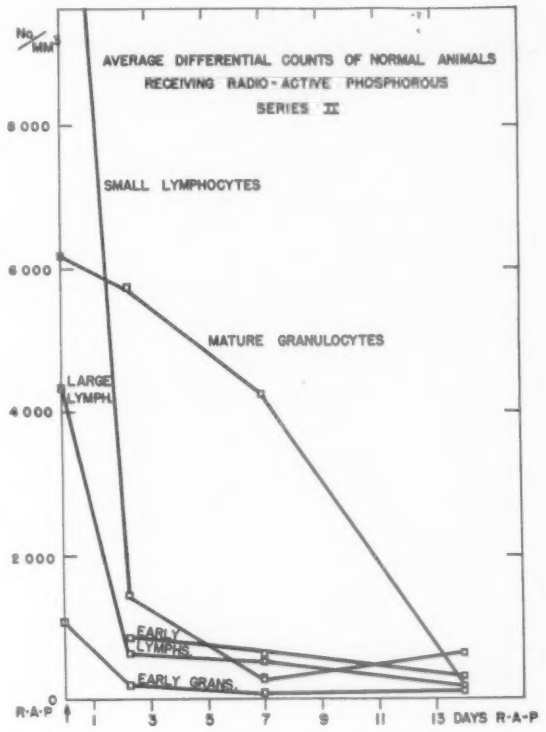

FIG. 2

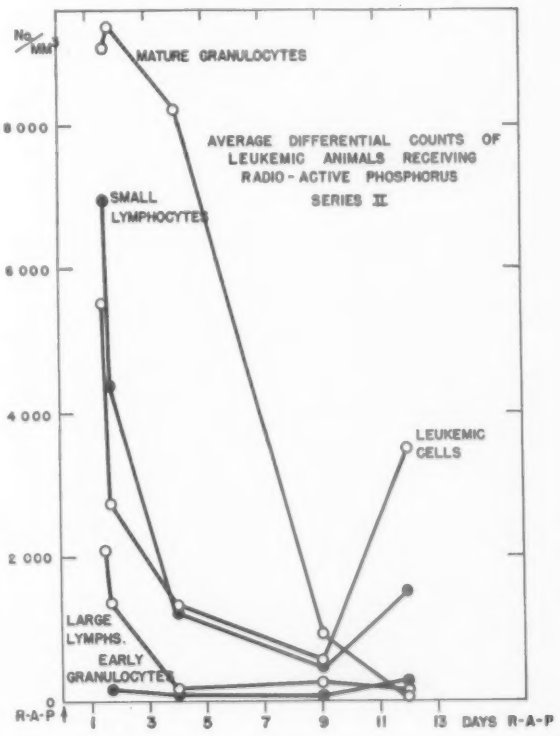

FIG. 3

FIG. 4. Normal mouse bone and marrow. ×70.

FIG. 5. Bone and marrow 214 hours after injection of 218 microcuries of radiophosphorus. Note aplastic appearance. There is a preponderance of mature and nucleated red blood cells. ×70.

have been purposely high in order to bring out the differential effects of P^{32}.

Histologic Studies of Tissues. In the bone marrow of normal mice treated with P^{32}, early granulocytes showed a slight increase sixty hours after P^{32} administration. A decrease followed which persisted for about five hundred hours when a slight increase was again noted. The megalokaryocytes

FIG. 6. Leukemic (twenty days) bone marrow. ×70.

were relatively unaffected. At sixty hours there was no increased vascularization, and at five hundred hours vascularization was decreased. The marrow presented an aplastic appearance, with red blood cells and early red blood cells forming the chief elements (see Fig. 4 and 5).

Eight days after injection with lymphoma cells the marrow appeared normal. As tissue infiltration with these cells progressed, there was a decrease in the number of early white blood cell forms and the megalokaryocytes disappeared. No significant change in vascularity was ob-

served. The lymphoma cells formed a solid mass at twenty days and occupied the entire marrow cavity (see Fig. 6).

When the leukemic mice were given P^{32} eight days after lymphoma injection, many normoblasts were present in the marrow six days after that. The megalokaryocytes remained normal in number and appear-

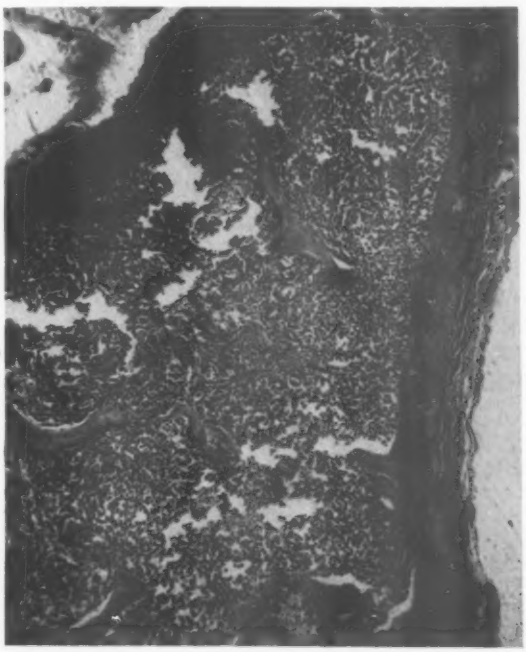

Fig. 7. Leukemic (twenty days) bone marrow 306 hours after injection of 218 microcuries of radiophosphorus. ×70.

ance until they disappeared. The granulocytes increased up to sixteen days after lymphoma injection and then decreased until at twenty days they were practically absent. No marked number of lymphoma cells were noted until twenty days, and even at that time the extent of the invasion was much less than that in the untreated animals (see Fig. 6 and 7).

In the mice receiving lymphoma, slight lymphomatous infiltration occurred in the liver after twelve days. At twenty days substantial perivascular infiltration with these cells was noted. In the terminal stages of the disease the blood vessels of the liver appeared to be dilated. The blood vessels

Fig. 8. Normal mouse liver. ×830.

of the livers of the lymphomatous animals showed definite dilatation eight days after P^{32} injection. No infiltration by lymphoma cells was observed until twenty days after lymphoma transmission. At that time and thereafter, the degree of infiltration appeared to be much less than in those

Fig. 9. Mouse liver 526 hours after injection of 218 microcuries of radiophosphorus. ×830.

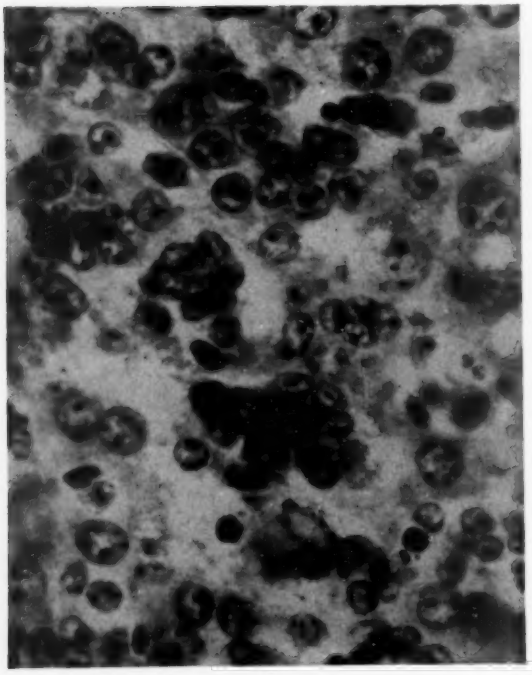

FIG. 10. Leukemic (twenty days) liver. ×830.

FIG. 11. Leukemic (twenty-two days) liver 360 hours after injection of 318 microcuries of radiophosphorus. ×830.

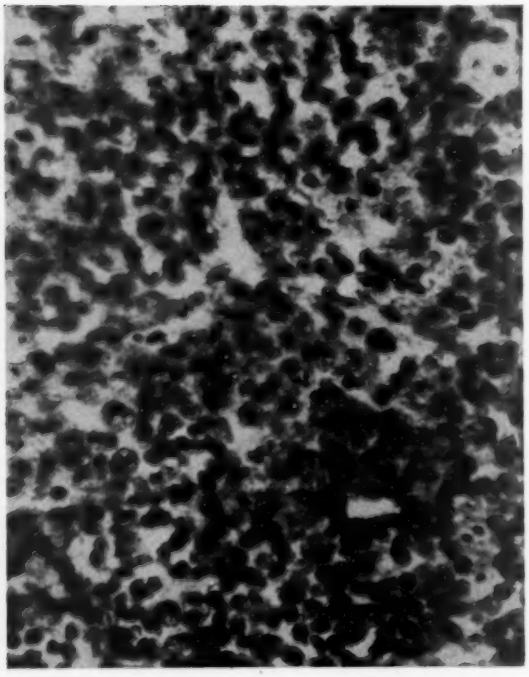

FIG. 12. Normal mouse lymph node. ×640.

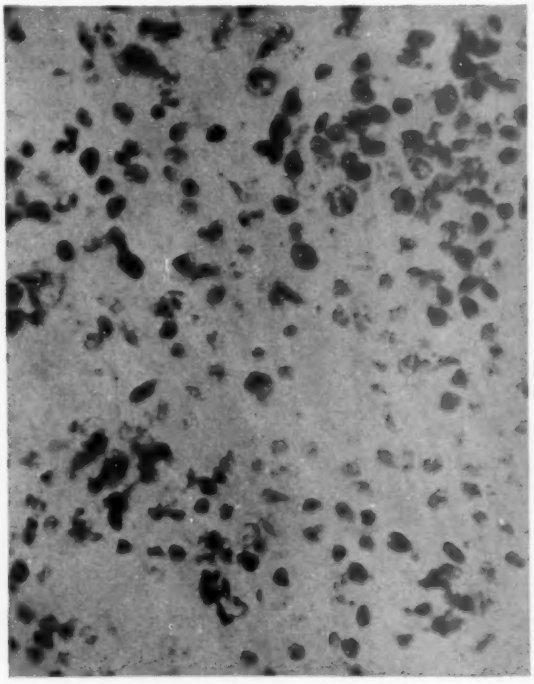

FIG. 13. Mouse lymph node 217 hours after injection with 195.3 microcuries of radiophosphorus. ×640.

lymphomatous animals which received no P^{32} (see Fig. 8, 9, 10 and 11).

The chief effect of P^{32} on the lymph nodes of normal mice was a decrease in gross size and cellularity; early granulocytes were frequent and the relative proportion of large lymphocytes increased. With respect to mice receiving only lymphoma cell injection, the lymph nodes became infiltrated and increased in size eight to sixteen days later. At twenty days the infiltration of lymph nodes by lymphoma cells was very obvious. When lymphomatous mice were given P^{32} eight days after lymphoma injection, no lymphoma cell infiltration was observed until nineteen days after the introduction of lymphoma cells (see Fig. 12, 13, 14 and 15).

Studies of the spleens of normal mice seventy hours after P^{32} administration showed some lymphocytic degeneration. There was an increase of red pulp with a proportional decrease of white pulp. The megalokaryocytes appeared normal. Two hundred hours after P^{32} administration the gross size of the spleen was greatly reduced. The large lymphocytes were predominant. The number of mitotic figures had decreased. Granulocytes were scarce and no megalokaryocytes could be observed.

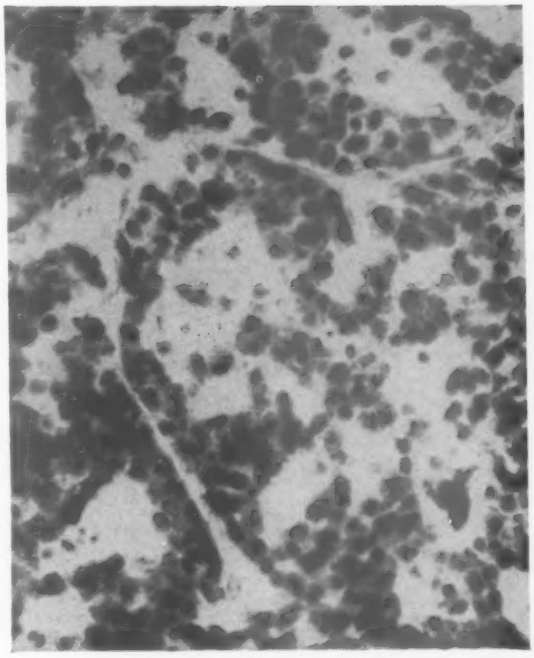

Fig. 15. Leukemic (twenty-two days) lymph node 360 hours after injection of 218 microcuries of radiophosphorus. Compare with Figure 14. ×640.

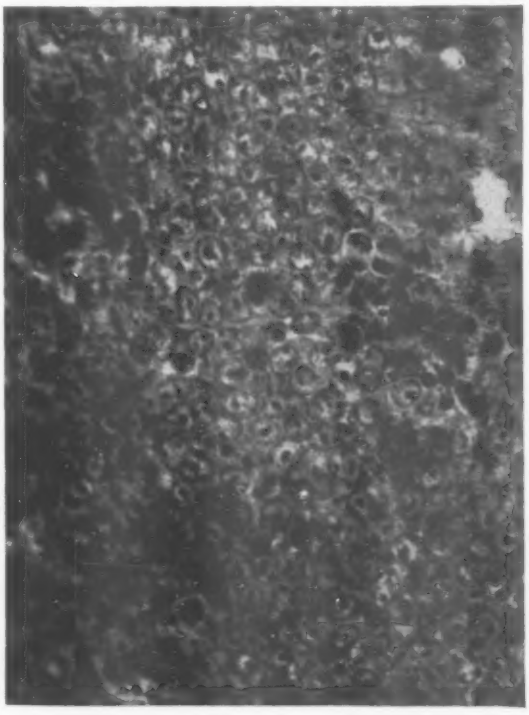

Fig. 14. Leukemic (twenty days) lymph node. Note dense leukemic cells. ×640.

When the mice were injected with lymphoma cells a large increase in the size of the spleen was observed twenty days later; this increase was due to invasion by lymphoma cells. Of the remaining normal lymphocytes, the large forms were predominant.

When the mice receiving lymphoma cells were given P^{32} eight days later, only slight splenic invasion by lymphoma cells was observed. At twenty days after lymphoma injection, infiltration with lymphoma cells was observed but was not as great as in the untreated leukemic animals (see Fig. 16, 17, 18 and 19).

Discussion of Histological and Hematological Results. These results show an initial depressing action of P^{32} on the lymphocyte

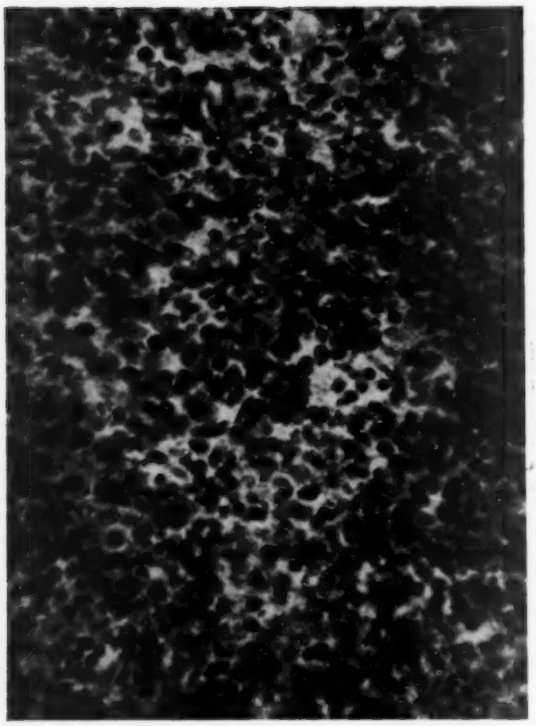

FIG. 16. Normal mouse spleen. ×330.

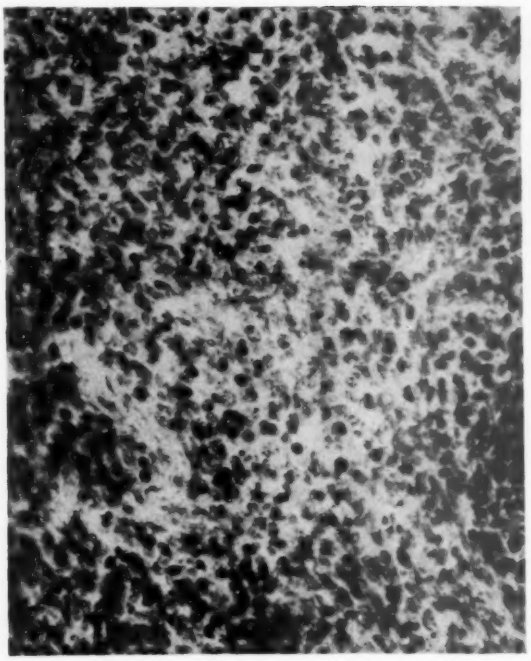

FIG. 17. Mouse spleen 214 hours after injection of 218 microcuries of radiophosphorus. Note decreased cellularity of tissue. ×330.

FIG. 18. Leukemic (twenty days) spleen. ×330.

FIG. 19. Leukemic (thirteen days) spleen 140 hours after injection with 218 microcuries of radiophosphorus. ×330.

series in the peripheral blood stream. After four days, when the granulocytes begin to drop off markedly, there already is an increasing deposition of P^{32} in bone. The retention of P^{32} by bone is of much longer duration than by lymphatic or any other tissue. It seems justifiable to correlate this fact with the more prolonged drop of the granulocytes. We have already noted that P^{32} seemed to have a greater initial depressing effect on the white cell count of the leukemic than on the normal animals, but there is no direct evidence that the neoplastic cells are more radiosensitive than other cells.

If the histological data on the treated and untreated lymphomatous animals are compared, we note that at corresponding stages in the lymphomatous process the treated lymphomatous animals have less infiltration of organs by leukemic cells than the non-treated groups.

We may conclude that when relatively large doses of P^{32} are given to normal and leukemic animals, the greatest drop in white blood cells occurs during the first four days after administration. The chief cell type to be affected is the lymphocyte and the lymphoma or leukemic cell. During the following five days the rate of drop is diminished, but during this period the greatest decrease in granulocytes occurs. Thereafter, there is a gradual increase in small lymphocytes and leukemic cells although the granulocytes continue to decrease in number. P^{32} delays the time of appearance of dense infiltration of tissues.

PART II

Experimental Therapy of Leukemic or Lymphomatous Mice. Studies of the exchange of P^{32} in leukemic tissues of mice revealed a higher uptake than in other soft tissues. This greater retention reached a maximum about three days after administration of P^{32}, but lymphomatous tissue retained more P^{32} as long as nineteen days after P^{32} administration when compared with normal lymph node, although this difference tended to decrease with the passage of time.

From a therapeutic point of view this greater uptake in neoplastic tissue is of interest since these cells receive relatively more irradiation. When mice inoculated with lymphoma were given single doses of P^{32}, greater than 75 microcuries, death always resulted. At postmortem it is difficult to determine whether the mice have died from lymphoma or from radiation effects. On the other hand, we know from previous work, that the lethal dose for normal mice of these ages and weights is about 75 microcuries. In the following experiments it was decided to give the active phosphorus in several smaller doses since by this method it was hoped that activities lethal to lymphoma but sublethal to the animal could be maintained for long periods of time.

Experiment 1. The mice used were "A" strain, eight week old males. They were given intraperitoneally a suspension of lymphoma cells. All twenty of the mice developed enlarged spleens, tumors, or both, eight days later. Ten mice were used as controls and all died with evidence of generalized lymphomatosis. The other 10 mice were given P^{32} intraperitoneally* as follows: eight days after lymphoma transmission, 27.8 microcuries; eleven days, 24.2 microcuries; fifteen days, 13 microcuries. Eight of these animals died. The cause of death was doubtful in some cases (i.e., from lymphoma or radiation effects). However, 2 of these animals which had shown definite evidence of lymphoma (enlarged palpable spleens) recovered. Their spleens were no longer palpable and they were alive five months after injection of lymphoma cells and in good condition.

Experiment 2. Twenty-five "A" strain males were given intraperitoneally a lymphoma cell suspension. All of these mice developed peritoneal tumors or large spleens or both within ten days. Fifteen mice were used as controls and 10 were treated with P^{32}. In this experiment the first dosage of P^{32} (20 microcuries) was given five days after lymphoma injection. At fifteen days, 28 microcuries, and at twenty-one days, 20 microcuries of additional P^{32} were given. All

* In this and following experiments P^{32} was administered intraperitoneally as an isotonic salt of sodium phosphate.

of the control animals died of generalized leukemia. Nine of the treated animals died. One of the treated animals recovered from the lymphoma after developing mesenteric tumors and an enlarged spleen. This animal was alive six months later and appeared to be in good health with no signs of lymphoma.

Experiment 3. Twenty-five "A" strain males were given intraperitoneally lymphoma cells. Twenty were selected as takes, as evidenced by enlarged spleens or palpable intraperitoneal tumors, and 10 of these were set aside as controls. The other 5 mice subsequently developed lymphomatosis and died but were not used in this experiment. All of the control mice died with the generalized disease with an average survival time of 23.4 days. All of the animals treated with radiophosphorus also died. (Average life 26.2 days.) The radiophosphorus was given as follows: 13.75 microcuries thirteen days after injection; 13.1 microcuries fourteen days and 25.5 microcuries seventeen days; total dosage 52.35 microcuries.

Experiment 4. Twenty-one Swiss "A"* crosses were given intraperitoneally a suspension of lymphoma cells. All of the animals developed leukemia from nine to twenty days later.

A. Nine days after injection, 8 of these mice were recognized as lymphoma "takes." Four were used as controls. Four received radiophosphorus as follows: eleven days after tumor transmission, 22 microcuries; twelve days, 17.5 microcuries; and seventeen days, 20 microcuries; a total of 59.5 microcuries. One mouse, which had a markedly enlarged spleen prior to P^{32} administration, was given an additional dose of 20 microcuries at twenty days, making the total dosage 79.5 microcuries. This large spleen was observed on repeated examinations but gradually decreased in size until it appeared to be normal twenty-six days after lymphoma implantation. This animal was still alive and apparently normal five months later.

B. Later, 9 more takes were observed and 5 of these were used as controls. The remaining 4 were treated with P^{32} as follows: fourteen days after implantation, 30 microcuries; seventeen days, 7.5 microcuries; nineteen days 7.5 microcuries and twenty-five days 32 microcuries; total dosage, 77 microcuries. All of the controls died of generalized involvement and some had large subcutaneous tumors. Three of the treated animals died showing evidence of leukemia and subcutaneous tumors. The remaining mouse, which had an easily palpable tumor mass when treatment was started, slowly recovered and five months later was in good condition and apparently free from lymphomatosis. The tumor was gradually resorbed.

The average survival time of the control mice in the entire experiment was 33.7 days, whereas survival time for the treated animals dying was 34.7 days.

Experiment 5. Twenty "A" strain mice were given lymphoma intraperitoneally. Ten controls died with a survival time of 21.8 days and the ten treated animals died with a survival time of 26.6 days. The radiophosphorus dosage was as follows: 20 microcuries thirteen days after, 2.9 microcuries seventeen days after, and 8 microcuries twenty-five days after implantation; total dosage, 30.9 microcuries.

To conclude, in the five therapeutic experiments described, regression resulted in three of them. The amount of P^{32} given varied in the different experiments, and regressions resulted when the total dosage was 65, 68, 77 and 79.5 microcuries, the time periods during which the P^{32} was administered varying from nine to sixteen days. There were regressions in 5 of 31 treated animals. Although the doses used in these experiments were sublethal, and no blood studies were carried out, it is certain that marked changes in the white and red blood cell counts occurred. In these experiments which were terminated because of the transference of the medical research activities of this laboratory to other problems in 1941, no further follow-up of the apparently cured mice was made. It is possible that their recovery (aided by the inhibitory effect of P^{32} on the neoplastic growth) from implanted lymphomatosis had conferred on them an immunity to further transplantation of this tumor into them.

Relation of These Results to the Clinical Use of P^{32}. These investigations in mice and others carried out on monkeys[14] demonstrate that with relatively large doses of P^{32}, the bone marrow can be wiped out.

* These were the offspring of matings of "A" strain and Swiss n.i e.

With sufficient doses, all elements including the platelets, white cells and red cells are affected. Thus in the treatment of leukemia with P^{32}, unless the leukemic white cells are more radiosensitive than the other elements in the marrow, it is not possible to prevent their rapid production, without at the same time inhibiting red blood cell and platelet formation.[8,4,7] It is true that rapidly growing cells such as leukemic cells take up more P^{32} than other cells, but if these cells are adjacent to other types of cells, the latter will also receive irradiation since the beta rays from P^{32} penetrate several millimeters in tissue. In the treatment of leukemia one nearly always eventually arrives at the point where the leukemic cells become radioresistant or at least no more radiosensitive than the platelet and red cell producing centers. Thus even though there is some selective irradiation, especially by virtue of P^{32} localization in infiltrated lymph nodes, spleen and liver, one eventually faces the same problem as is faced when using spray or local roentgen irradiation since the whole marrow, and therefore both leukemic and normal elements, are being irradiated. These predominant effects of P^{32} on the various elements of the marrow constitute the limiting factor in its use in the therapy of leukemia and make its use of doubtful value in lymphosarcoma and allied diseases. Recent investigations in this laboratory indicate that it may be possible, however, to cause a relatively greater deposition of P^{32} in the tissues other than the bone marrow and thus decrease the bone marrow irradiation.

In the general problem of the radiation therapy of neoplastic disease (and leukemia may well not be a neoplasm), one should find a radio element or compound of the element which would localize to a high degree in or immediately around the neoplastic cell. There are examples of such localization in normal tissue in the case of radioactive iodine in the thyroid gland[3] and chromic phosphate (radioactive phosphorus)[5] in the liver and spleen. In these two instances it has been possible to remove in animals the thyroids and spleens by selective irradiation without serious damage to the rest of the body. These are examples of selective irradiation in the true sense of the word. Since leukemia is such a diffuse disease, the possibilities of finding a method of true selective irradiation are not great, and for this and other reasons we must look for the control of this disease by some method other than irradiation.

SUMMARY

Hematological and histological studies on normal and lymphomatous mice which have been given radiophosphorus revealed characteristic effects on the hematopoietic tissues. Although a few animals recovered from generalized lymphomatosis after treatment with P^{32}, no evidence of any increased radiosensitivity of the neoplastic cells was observed. The limiting factors in the use of roentgen irradiation and P^{32} in the therapy of leukemia and allied diseases are discussed.

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STUDIES ON THE EFFECTS OF RADIOACTIVE SODIUM AND OF ROENTGEN RAYS ON NORMAL AND LEUKEMIC MICE*

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THE possibility of employing artificially produced radioactive substances in therapy is attractive, and various trials of some of them have been made.[1,2] In general, it has been considered desirable to use a substance which soon after administration is selectively concentrated in certain organs. Thus radio-iodine is largely localized in the thyroid gland, radiostrontium in bone, radiophosphorus in bone, bone marrow, leukemic tissue and any rapidly growing cells. Because of this property of radiophosphorus, it has been administered in the treatment of leukemia and allied diseases, with some success.[2,3]

This work with radioactive phosphorus is still in the experimental stage and methods of administration are tentative. The results compare favorably with those of other methods (roentgen rays and arsenic) but no great improvement has as yet been claimed. It seems that until more definite conclusions can be drawn regarding superiority of radioactive phosphorus over roentgen irradiation, it is also worth while to investigate the effects of other radioactive substances which may have some advantages.

Radioactive sodium, at the present time the most readily prepared of all these isotopes, does not concentrate in any organ or group of organs, but instead is, within a short time after its administration, distributed throughout the extracellular fluids of the body. It emits penetrating beta and gamma rays, and thus, by virtue of its distribution, can administer a rather uniform irradiation to the entire body. Its half-life, 14.8 hours, is long enough to be useful and yet short enough so that dosage can be closely correlated with effect. Reaction to the administration of a therapeutic dose can be observed within a few days and further treatment can be regulated accordingly. Since sodium is a normal constituent of body fluids, and the radioactive isotope is administered as a small amount of isotonic saline, no physiological disturbance should occur as a result of its use, except such as would be produced by the action of the radiation. It might be expected to be valuable as a substitute or supplement for other types of irradiation in the treatment of leukemia and allied diseases.

Before using the material on human patients, it appeared desirable to study its effects on animals. Mice are particularly suitable because strains are available which exhibit a high spontaneous incidence of a condition similar to leukemia, presenting an abnormally high white blood count and enlarged lymph nodes. Accordingly, studies were undertaken with normal and leukemic mice of one of these strains, and normals of a strain which does not develop this condition. The results of the administration of radioactive sodium were compared to those of whole body roentgen irradiation. The effects to be reported at the present time are changes in blood counts and reduction in size of involved lymph nodes. Data are also presented regarding distribution of the radiosodium in various tissues and organs. A study of histologic effects is in progress and will be reported at a later date.

MATERIALS AND METHODS

The radioactive sodium, Na^{24}, was prepared by the Cyclotron Laboratory of the Physics

* With the aid of a grant from the Lilla Babbit Hyde Foundation. Presented at the Joint Meeting of the American Roentgen Ray Society and the Radiological Society of North America, Chicago, Ill., Sept. 24–29, 1944.

Department of Columbia University; it is a pleasure to acknowledge the cooperation of this group. A measured number of microcuries (μc.) of the material, in approximately isotonic sodium chloride, was injected subcutaneously in the flanks of the animal. Following such injection, the material is very rapidly distributed through all the extracellular body fluids. The gamma-ray activity of the animal was determined with a Geiger counter immediately after injection, and at intervals thereafter. The difference between the measured decrease in activity and the theoretical decrease due to radioactive decay represented the amount of material eliminated.

The roentgen rays were generated at 184 kv. (peak), with a filtration of 4 mm. Cu+2 mm. Al; at a distance of 40 cm., the dosage rate was 10 roentgens per minute. During the irradiation the animals were confined in a box made of plastic material. With radioactive sodium of half-life 14.8 hours, two-thirds of the dose of radiation is delivered in the first twenty-four hours, 22 per cent in the second twenty-four, and so on. Since the object of the experiments was to make a reasonable comparison between the effects of this material and of roentgen rays, the dose of the latter was divided, 70 per cent being given the first day and the remainder twenty-four hours later. Of course this division is not strictly comparable with the continuous irradiation from the sodium, but it was felt to offer a better approximation to it than administering the entire roentgen-ray dose at one sitting.

Mice of the Swiss and Ak strains were employed. The Swiss strain has been widely used for experimental studies; these were normal animals from Rockland Farms. The Ak strain, in the colony of the Crocker Laboratory (originally obtained from Dr. J. Furth, of Cornell Medical School), has a considerable incidence of spontaneous leukemia. Mrs. Mary Long, of that laboratory, supplied normal appearing mice of this strain and individuals with varying types of the disease. Blood for counts was taken from the tail. Micropipettes were used and care was taken to stop the loss of blood after samples were removed.

TABLE I

EFFECTS OF RADIOACTIVE SODIUM ON SWISS MICE

Dosage (μc.)	μc.gram (average)	Number of Individuals	Minimum Individual White Blood Count	Duration of Leukopenia Days	Duration of Erythropenia Days	Average Survival Days
0	0	5	8,500	0	0	25+
150	5.55	3	6,216	0	0	25+
300	10.0	5	3,100	1	0	25+
500	14.7	1	3,850	3	0	25+
1,000	38.5	9	4,150	6	0	25+
1,500	47.0	2	1,100	15	2	25+
2,000	71.5	2	350	15	1	25+
2,500	83.5	3	200	14	0*	20
3,000	100.0	1	150	11+*	0*	12
4,000	125.0	1	425	7+*	0*	8

* Does not represent total injury as animal died before the end of the experiment.

EXPERIMENTAL RESULTS

1. *Effects on Normal Swiss Mice of Different Doses of Radioactive Sodium.* Twenty-seven animals were injected with amounts of radioactive sodium varying from 150 to 4,000 microcuries (5.55 to 125 microcuries per gram of body weight). Survival time, and white and red blood counts for this group are summarized in Table 1. Controls consisted of 2 non-injected animals, 2 animals injected with isotonic non-radioactive saline, and one injected with a sample of the originally radioactive material, after its activity had decayed to a negligible amount. In no case did any of these animals exhibit any unusual change in its blood count.

The treated animals were sacrificed twenty-five days after the injection of the radiosodium, if they survived that long. Doses of 3,000 and 4,000 microcuries were fatal in less than two weeks, and of the 3 animals receiving 2,500 microcuries, 1 died in ten days. A temporary reduction in leukocyte count was produced by a dose as low as 300 microcuries. As indicated in the table, with increase in dosage the minimum individual white blood count was lowered; the duration of leukopenia* was increased, to a maximum of about fifteen days, and the average survival time was diminished. Erythropenia occurred only after relatively large doses, and does not appear as soon as leukopenia.

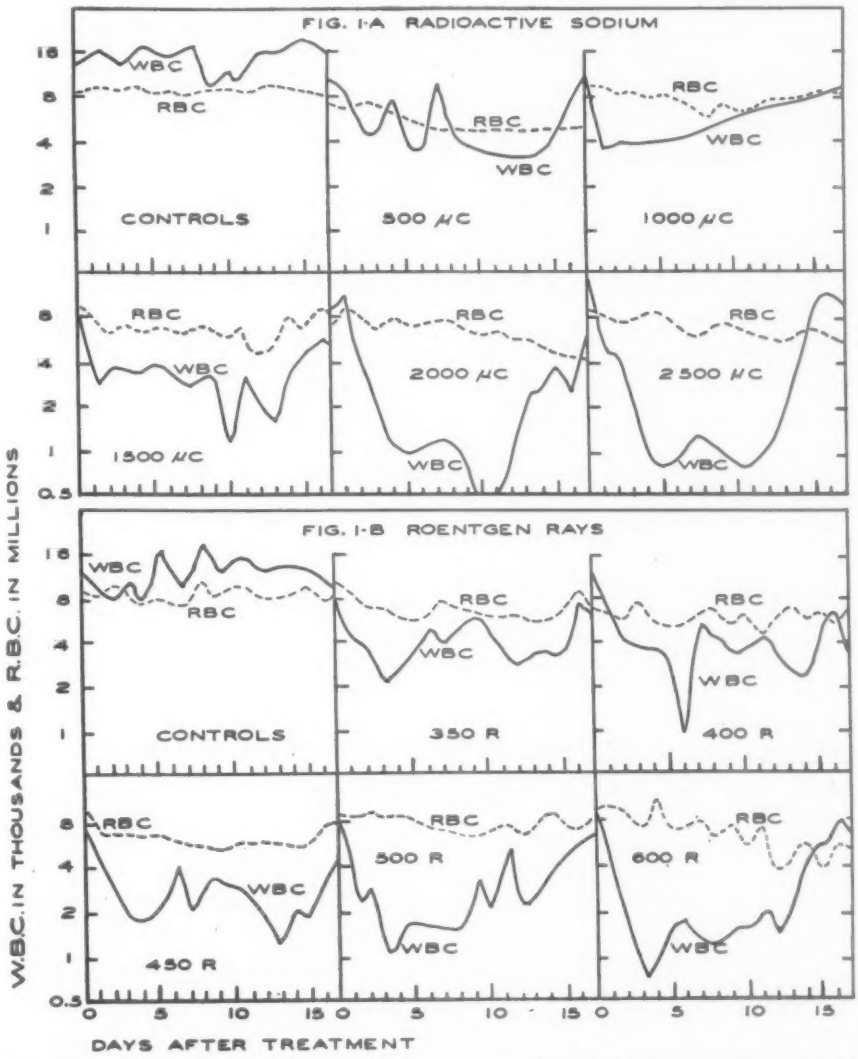

FIG. 1. *A*, changes in the erythrocyte and leukocyte counts of mice following treatment with radioactive sodium. *B*, changes in erythrocyte and leukocyte counts following roentgen irradiation. Daily determinations were alternated between two animals of each graph. For further details see text and Tables I and II.

The average daily white and red blood counts of these animals are shown in Figure

* For purposes of this study, a white blood count of 5,000 or below is called leukopenia; a red blood count of 5,000,000 or below is called erythropenia.

1*A*. Since the individual fluctuations are great, a logarithmic scale has been adopted to smooth out small variations and emphasize significant changes. The increase in degree and duration of leukopenia with increased dose is evident, as is also the fact that in animals of this strain considerable depression in the white blood count may be produced with no noticeable effect on the red blood count during this period.

2. *Effects on Normal Swiss Mice of Different Doses of Roentgen Rays*. Nineteen animals were exposed to doses of from 350 to 1,000 r of roentgen rays, 70 per cent of the dose administered one day and 30 per cent the next; the results are shown in Table II and Figure 1*B*. The curves of Figure 1*B* closely resemble those of Figure 1*A*, indicating that the effects of the two treatments are of the same kind. From Table II it is also evident that as the dosage is increased the leukopenia becomes more pronounced and prolonged, to a maximum duration of about fifteen days, erythrocytes are reduced in number, and finally survival is affected.

TABLE II

EFFECTS OF ROENTGEN IRRADIATION ON SWISS MICE

Dosage (Roentgens)	Number of Individuals	Minimum Individual White Blood Count	Duration of Luekopenia Days	Duration of Erythropenia Days	Average Survival Days
0	2	8,000	0	0	25+
350	2	2,000	8	0	25+
400	2	1,000	9	1	25+
450	2	1,250	13	2	25+
500	2	900	14	0	25+
600	2	650	15	2	25+
700	2	500	15	4	25+
800	3	250	15	4	11
900	3	300	15	4	16
1,000	1	300	Died at 5 days		5

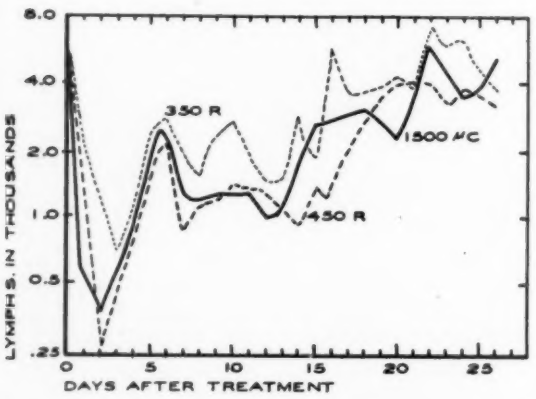

FIG. 2. Comparison of changes in leukocyte count produced by 1,500 microcuries of radiosodium (solid line) and by 350 and 450 roentgens of roentgen rays (dotted and dashed lines). See text for further details.

Not only do radiosodium and roentgen rays have similar effects on total white and red blood counts, but they also produce similar changes in the differential counts. Lymphocytes are more radiosensitive than the other leukocytes; typical effects are shown in Figure 2. The percentage of lymphocytes was multiplied by the total number of leukocytes for that day to obtain the number of cells of that type per cubic millimeter of blood. At this dosage level either sort of irradiation produced a lymphopenia of approximately two weeks' duration. A minimum was reached within three days, followed by recovery. The heavier dosage (450 r) of roentgen radiation produced a lower minimum and slower recovery than did the 350 r treatment. The effects of 1,500 μc. appear to be more nearly like those of the 450 r experiment.

Table III

EFFECTS OF RADIOACTIVE SODIUM ON AK MICE WITHOUT LYMPHADENOPATHY

Dose μc.	Microcuries per gm.	Initial White Blood Count	Minimum White Blood Count	Duration of Leukopenia Days	Duration of Erythropenia Days	Survival Time Days
0	0	11,250	7,150	0	0	25+
74	2.2	9,650	10,450	0	0	25+
230	10.5	8,400	4,300	2	0	25+
390	16	10,000	4,900	3	0	25+
570	17	13,800	4,250	5	0	25+
1,300	52	9,450	1,750	14	0	25+
3,000	94	25,300	300	6*	0*	8

* Does not indicate maximum effect as animal died before end of experiment.

3. *Effects of Radioactive Sodium on Mice of a Leukemic Strain.*

A. Ak animals without lymphadenopathy: Before working with Ak animals with adenopathy, it was desirable to study the effects produced by radiosodium in apparently normal mice of this strain. Seven Ak animals with normal white counts and without enlarged nodes were given the same series of doses as the normal Swiss mice. The results, given in Table III show effects similar to those in Table I. It appears that any difference in radiosensitivity between these animals and those of the same strain with lymphadenopathy may be attributed to their disease.

B. Ak animals with aleukemic adenopathy: From time to time animals with palpable nodes were found in the Ak colony. Blood counts were made on them, and those with white counts higher than 30,000

Table IV

EFFECTS OF RADIOACTIVE SODIUM ON AK MICE WITH ALEUKEMIC ADENOPATHY

Animal	Dose μc.	Microcuries per gm.	Initial White Blood Count	Minimum White Blood Count	Duration of Leukopenia Days	Duration of Erythropenia Days	Survival Time Days	Nodes 0 day	Nodes 1 week	Nodes 2 weeks
Ak35	0	0	10,200	4,000	1	0	14+	Medium	Medium	Medium
Ak33	0	0	8,700	6,300	0	1	14+	Small	Small	Small
Ak3	150	6	12,350	10,800	0	0	14+	Medium	Small	Very small
Ak2	146	5	15,500	9,500	0	0	14+	Medium	Small	None
Ak15	170	5	23,150	11,600	0	0	14+	Medium	Very small	Very small
Ak5	170	5	9,650	13,400	0	0	14+	Medium	Small	Small
Ak6	164	5	11,900	8,100	0	0	14+	Medium	Small	None
Ak10	345	13	9,830	3,000	2	0	14+	Very small	None	None
Ak27	514	14	23,250	3,600	3	0	14+	Very small	None	None
Ak29	510	17	25,850	4,500	1	3	14+	Small	None	Very small
Ak11	660	21	16,800	3,800	2	0	14+	Very small	None	Very small
Ak34	800	28	22,500	2,950	2	0	14+	Large	None	Very small
Ak12	1,000	29	16,950	3,000	1	0	14+	Small	None	Small
Ak26	1,014	30	25,350	3,750	3	0	14+	Medium	Very small	Small
Ak22	920	32	12,250	1,700	2	0	14+	Medium	Small	None
Ak8	1,250	40	7,550	1,800	14	1	14+	Large	Small	Small
Ak31	1,450	68	11,350	1,650	3	0	14+	Large	Very small	None
Ak13	3,000	94	11,800	350	4(death)	4(death)	4	Large	Small	—

were considered leukemic, those with lower counts, aleukemic. Eighteen animals of the latter type have been given radiosodium; the results are shown in Table IV. The minimum white blood counts corresponded roughly to those of normal animals receiving the same doses. The duration of leukopenia was the same or slightly less than in some of the mice received additional treatment and were no longer suitable for the comparison with singly treated normals.) Doses of the order of 20 microcuries per gram produced marked leukopenia, usually of rather short duration. The white count recovered rapidly to normal limits, but rarely exceeded the maximum normal level

TABLE V

EFFECTS OF RADIOACTIVE SODIUM ON AK MICE WITH LEUKEMIC ADENOPATHY

Animal	Dose μc.	Microcuries per gm.	Initial White Blood Count	Minimum White Blood Count	Duration Aleukemia Days	Duration Leukopenia Days	Duration Erythropenia Days	Survival Time Days	Nodes 0 day	Nodes 1 week	Nodes 2 weeks
Ak17	0	0	50,000	32,000	2	0	0	14+	Medium	Large	Very large
Ak23	0	0	28,500	21,700	1	0	1	14+	Large	Large	Very large
Ak39	500	14	77,000	17,250	4	0	1	14+	Very large	Medium	Very large
Ak36	500	19	141,500	2,000	8	1	10	14+	Large	Very small	Very small
Ak7	590	20	32,000	1,400	14	3	0	14+	Large	Small	Small
Ak25	604	21	38,750	4,250	14	0	0	14+	Very small	None	None
Ak24	651	24	55,000	5,250	14	1	3	14+	Small	None	None
Ak20	990	32	38,300	600	14	11	0	14+	Large	Medium	Very small
Ak30	1,444	51	46,800	1,150	14	11	10	14+	Large	Very small	Very small
Ak19	1,767	58	42,100	100	10	8	1	11	Large	None	Very small
Ak16	3,000	100	53,750	200	12	11	10	12	Large	None	None

the normals. The lymph nodes responded, at least temporarily, to doses so small as to have no apparent effect on the blood count. The effect on the nodes naturally became more pronounced as dosage increased.

C. Ak animals with leukemic adenopathy. Eleven mice had palpable nodes and white blood counts above 30,000. Some of these had low red blood counts and were weak. All animals in this group were very sensitive to the radiation, as shown by the results in Table V. (Since the initial response to a single dose of radiation was the principal point of this study, detailed data are given for only fourteen days. After that, (30,000) during the fourteen day period. Erythropenia occurred more frequently and with lower doses than in the case of any non-leukemic group. Decrease in size of nodes was marked.

There was less uniformity in response of these animals than in the others. Figure 3 shows lymphocyte and polymorphonuclear* counts for 6 mice receiving approximately the same amounts of irradiation (see Tables IV and V for details). All had palpable nodes. Animals Ak36, 39 and 25 had unusually high lymphocyte counts. The number of polymorphonuclears and

* Includes all granulocytes.

lymphocytes of Ak27 were within control limits. Animals Ak29 and 24 had high polymorphonuclear counts. In general, they show more reduction in lymphocyte number than was produced by comparable amounts of irradiation in control animals. In animals having a high lymphocyte count (Ak39, Ak36) lymphopenia developed rapidly but did not persist as long as it did in animals Ak25, Ak27, with a more nearly normal initial lymphocyte count. Animals Ak29 and Ak24 had a condition similar to myeloid leukemia. Although the polymorphonuclears were affected more than those of control animals, the differential susceptibility was not as evident as that of hyperplastic lymphocytes. The susceptibility of the blood cells apparently was related to the character of the disease and the physical condition of the animal. Ak36 was the only animal in this group whose erythrocytes were reduced in number following treatment. This animal was weak at the time of treatment and it is difficult to determine whether the anemia resulted from the irradiation or was due to continuation of the disease.

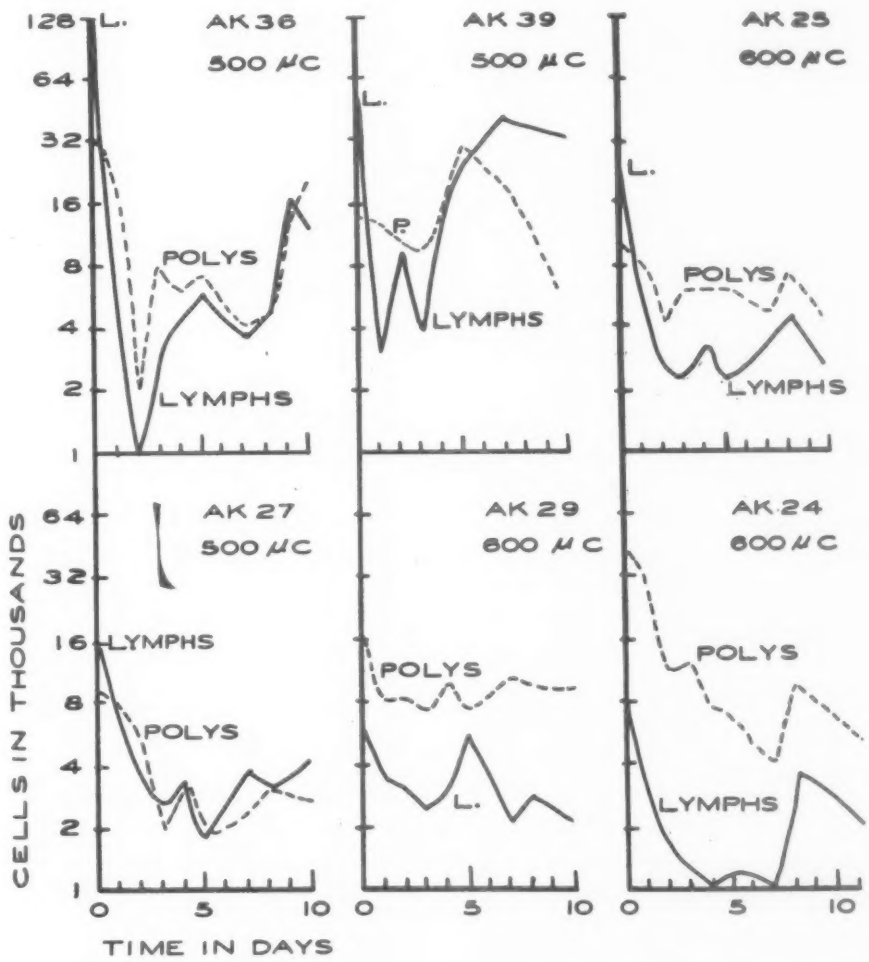

FIG. 3. Effects of radioactive sodium on blood counts of Ak animals with nodes. Solid line indicates number of lymphocytes and broken line represents number of polymorphonuclears. For further details see text and Tables III and IV.

4. *Elimination.* Elimination was studied

by measuring with a Geiger counter the gamma-ray activity of the animal immediately after injection, and at various times within the next seventy-two hours. The activity at any time was expressed as a percentage of the initial activity, and the values plotted as shown in Figure 4*A*. The time more than an hour or two after the injection, the activity of the animal as registered by the counter was less than the theoretical amount; this is due to excretion of some sodium by the kidneys and possibly by other routes. Although there was considerable variation among individ-

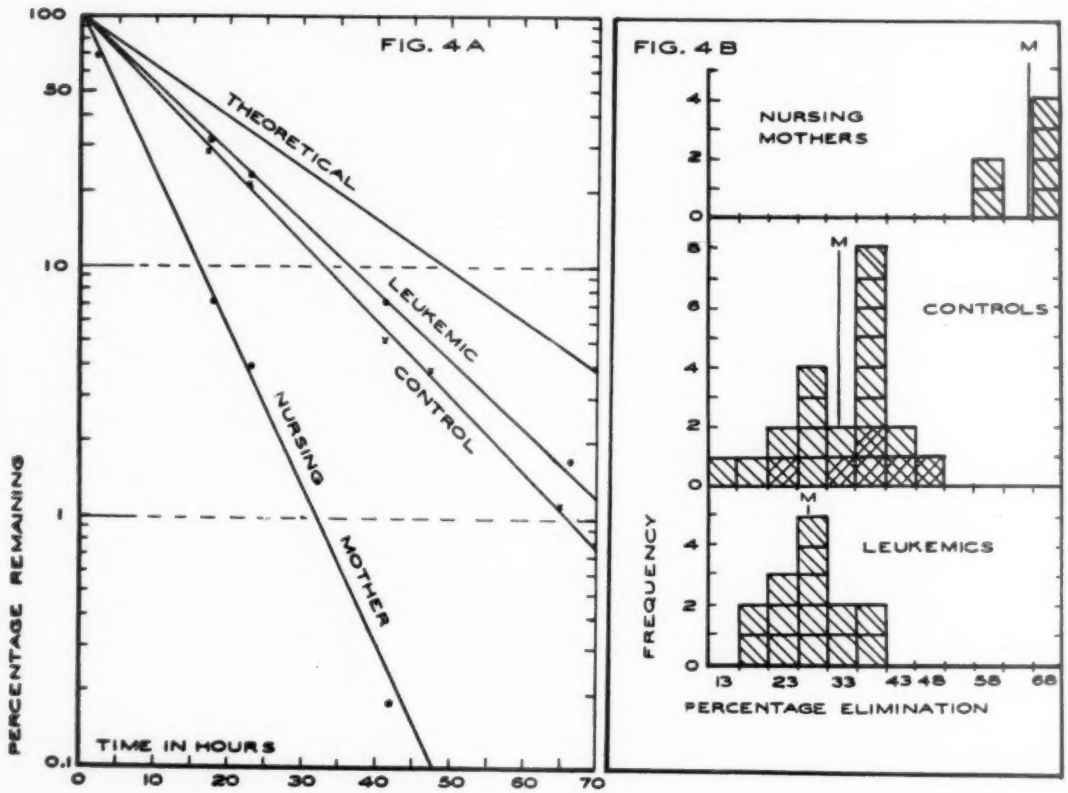

FIG. 4. *A*, graph showing radioactivity remaining in animals at different times following administration of radiosodium. Method of determining percentage elimination from these curves is explained in text. *B*, frequency graph showing individual animals grouped according to percentage elimination. Abscissa indicates percentage elimination in increments of 5 per cent. Ordinate shows number of cases in each class. In chart for controls, Swiss strain mice are indicated by singly cross-hatched blocks, non-leukemic Ak animals by doubly cross-hatched.

upper line (theoretical) is calculated from the known radioactive decay rate of Na^{24}; from time to time the material used has been checked against this curve to be sure there was no contamination with other radioactive substances. If the animals did not eliminate any of the administered material, their activity should also follow this curve. However, it was found that at any

ual animals, the points for the activity of any one at different times lie essentially on a straight line, when plotted to a logarithmic scale.

In Figure 4*A* are shown samples of such curves, for 1 control and 1 leukemic animal, and, for contrast, a nursing mother mouse of the Ak strain. It is impracticable to present these curves for each animal, since

those for controls and leukemics all lie together within a fairly narrow region which is definitely below the theoretical curve, indicating that appreciable elimination takes place. Due to this elimination, the effective dose of radiosodium is less than the administered amount. An approximation to the amount excreted can be obtained in the following manner: Any radioactive element has an "average life" which is 1.43 times its half-life. The total activity of 1 microcurie of the element throughout its entire lifetime, with its regularly diminishing strength, is the same as if it continued at full strength for its average life and then suddenly ceased to exist. The half-life of radiosodium is 14.8 hours; its average life is $14.8 \times 1.43 = 21.2$ hours. Hence in its total disintegration its energy can be expressed by 21.2 microcurie-hours per initial microcurie. Since the curves for the experimental animals are linear when plotted in the same manner as the theoretical one (Fig. 4*A*), they can be handled in the same manner as pure radioactivity curves; the situation is *as if* an element were being used which had a somewhat shorter life than sodium and was not eliminated at all. By plotting the curve for each animal to a large scale, the *effective* half-life for radioactivity in the body can be determined, and from these values the effective average lives can be calculated. The elimination is then equivalent to the difference between the theoretical and the effective average lives. For instance, the control mouse shown in Figure 4*A* had an effective half-life of 10 hours or an effective average life of 14.3 hours. Then instead of 21.2 microcurie-hours of irradiation per microcurie, this animal received 14.3 microcurie-hours. This means a loss by elimination of the effect of $21.2 - 14.3 = 6.9$ microcurie-hours per microcurie; $(6.9/21.2) = 33$ per cent.

Since there were relatively few animals in the experiment and considerable individual variation in the elimination percentage, average values are of little significance. Therefore a frequency graph was constructed (Fig. 4*B*) in order to compare data on this subject. The animals were grouped in steps differing from each other by 5 points (10–14, 15–19, etc.); the value for the mean of each group appears on the chart. Casual inspection leads to the conclusion that, on the average, the leukemics did not eliminate quite as much as the controls; however, the difference is found to be not statistically significant. Nursing mothers, on the other hand, do exhibit a significantly greater elimination, as might be expected, since these animals secrete sodium in the milk as well as in the urine. The radioactive sodium could be detected in the suckling babes within an hour of the time it was administered to the mother. This finding (that administered sodium is rapidly secreted in the milk) is in agreement with results obtained with humans.[6]

5. *Distribution of Radioactive Sodium in Various Organs and Tissues.* Several animals were sacrificed at various times after injection, and the gamma-ray radioactivity determined for various organs per gram of wet weight. Gamma-ray measurements were made in order to eliminate differences due to absorption of beta rays within the tissues when the samples were of different sizes. For convenience all data were related to the activity of the liver as unity. The results are shown in Table VI. At the end of two hours, as would be expected, the greatest activity was found in organs rich in blood or blood vessels. Later, as the material was more uniformly distributed through the body fluids and started to enter the cells, the differences were less marked.[7] No effort was made to squeeze the fluid out of the specimens, and the presence of varying amounts may have influenced the results considerably. It is, however, apparent that there was no considerable concentration of the material in any particular organ or tissue.

DISCUSSION

The results thus far presented indicate that the effects produced by subcutaneous injection of radioactive sodium are of the

same sort as those resulting from whole body roentgen irradiation. The physiological changes are more marked with higher doses, and the response is essentially the same in normal mice of the two strains which were tested.

On a basis of the series of normal animals treated with radioactive sodium and with roentgen rays, it is possible to estimate the number of microcuries of the sodium which produce the same general effect as 100 r of roentgen rays.

By inspection of the curves of Figures 1 and 2, it appears that 47 microcuries of radioactive sodium per gram of body weight produced about the same effect as 450 r of roentgen rays administered under the conditions of the experiment. This gives an equivalence of 10 microcuries per gram (neglecting elimination) and 100 r, which appears to hold within the dosage ranges studied, not only for effect on total white blood count but also on differential counts.

Marinelli[4] has published a method for determining the "equivalent roentgens" due to beta radiation delivered and completely absorbed within tissues, from radioactive substances introduced into the body and remaining there for total disintegration. While Na^{24} emits both beta and gamma rays, the latter are so penetrating that they escape from the small body of the mouse without giving up an appreciable portion of their energy, so their contribution to the physiological dose may be considered negligible. Some of the beta rays also escape from the body. An estimate of the amount thus lost can be made as follows: These beta rays are largely absorbed by 3 mm. of water (or tissue). Hence it may be assumed that all such rays originating more than 3 mm. deep within the animal will be completely absorbed; of those originating between 1.5 and 3 mm., about three-fourths will be absorbed, and of those originating within the first 1.5 mm., about one-half. If a 30 gm. mouse be regarded as an ellipsoid

TABLE VI

DISTRIBUTION OF RADIOACTIVE SODIUM IN VARIOUS TISSUES
RELATIVE ACTIVITY PER GRAM WET WEIGHT

Animal	Microcuries Injected	Hours after Injection	Blood	Kidney	Skin	Spleen	Liver	Gastrointestinal Tract	Lungs	Lymph Nodes	Sternum	Testicles	Muscle
(WX3; 2) Swiss	200	2.5	2.0	2.1	1.0		1.0	1.1					1.2*
(Na1, B3) Ak, no nodes	19	2		2.1	2.8	4.5	1.0					1.7	
(Ak 45) +nodes	30	2	2.5	2.3	4.7	.9	1.0	1.9	1.36	3.72	2.4		0.9
(WX3; 5, 6, 7, 8) Swiss	126	22	2.6	1.5		1.3	1.0	1.2	2.15		2.0	1.4	
(Na42) Swiss	500	22	1.8	1.7	1.16	1.04	1.0	1.22	1.17		1.0	0.9	0.6
(Na1, B1) Ak, no nodes	19	24	2.0	0.8	1.1	2.16	1.0					0.5	
(Ak14) Ak +nodes	3,110	24		1.0	1.0	1.0	1.0	1.2		1.2		1.1	
(Na43) Swiss	500	48	1.9	2.1	1.3		1.0	1.4	1.5				0.5
(Ak9) Ak +nodes	337	48			1.8	1.6	1.0			2.3			
(Ak37) Ak +nodes	500	45	2.7	2.0	2.2	0.8	1.0	1.4	1.1	0.8	1.87		0.6

* Thigh, +femur, etc.

of appropriate dimensions, a calculation on this basis leads to the conclusion that only about 75 per cent of the administered beta radiation is effective.

Using Marinelli's formula, it is found that with total absorption of beta rays and no absorption of gamma rays, a dose of 1 microcurie per gram of body weight of an animal should give 32 "equivalent roentgens." The biological effect would, however, be reduced to 75 per cent of this by the escape of some beta rays from the body and further to about 65 per cent of those that would be absorbed, by physiological elimination. Thus the 32 "equivalent roentgens" would be reduced to $32 \times 0.75 \times 0.65$ or 15.6. Then 10 microcuries per gram should deliver 156 "roentgens."

This is somewhat higher than the equivalence of 10 microcuries per gram to 100 roentgens deduced from the experimental data. However, the rates of administration of radiation are quite different, and the equivalence is admittedly rough. In view of all the approximations involved in both estimates, the discrepancy is not unreasonable. It indicates that the correspondence set up is not far wrong, but that there is need for both better experimental comparison and a more accurate method of deciding the portion of the theoretical dose actually effective within the animal.

The leukemic mice were more radiosensitive to the radioactive sodium than were the normals; they were not tested with roentgen rays. Their leukocyte count was reduced more rapidly than in the normals, but also recovery began to take place earlier. The question arises as to whether this is a true difference in response, or whether these mice retain more of the material, either because of less elimination, or concentration in lymph nodes, or both. In Figure 4 it is indicated that elimination in leukemics is not significantly less than in the controls. From Table VI it is evident that there is no marked concentration in lymph nodes. It is probable that, on the average, these animals, not being in as good general condition as the normals, are more readily affected by any injurious agent, but this is hardly sufficient to explain the difference in time of showing damage and time of recovery in the two groups. The leukemic cells apparently have a different radiosensitivity from the normal white cells, because of their rapid production and their relatively undifferentiated condition.

Some of these leukemic animals with greatly enlarged nodes appeared to have a disease comparable to acute leukemia in the human. They were usually weak at the beginning of treatment and although their condition showed temporary improvement, relapse occurred fairly soon and death ensued. Necropsy usually revealed multiple nodes and leukemic infiltration of other organs. In a few cases, such as Ak36, the animal was grossly free of leukemic disease but was anemic.

Some (more chronic) cases have been treated repeatedly with small doses of radiosodium at weekly or monthly intervals, and a considerable percentage of these have been kept alive and in fairly good condition for six months or more. There is, however, not yet sufficient data for a report on these cases.

The results obtained in this work do not lead directly to the formulation of a method for treating human leukemia with radioactive sodium. They do indicate that by its use a generalized radiation can be administered conveniently, in effective amounts.

It must be emphasized that the equivalence of 10 microcuries administered per gram of body weight to 100 roentgens is applicable only to mice, not to larger animals or to humans. In man, this amount would give a much higher dose of radiation, for two reasons. In the first place, whereas in the mouse practically no gamma radiation is effective, and only about three-fourths of the beta radiation, in the human practically all of the beta rays and a considerable portion of the gamma rays are absorbed. Furthermore, the mouse eliminates about a third of the material in three days, while in

this period the average human excretes less than 10 per cent.[5] It is safe to estimate that the "equivalent roentgens" from a given dose *per gram of body weight* in the human would be at least twice as much as in a mouse.

SUMMARY

The effects of radioactive sodium and of whole body roentgen irradiation on white mice have been compared. Results in reduction of white and red blood counts and in shortening of life are similar for the two types of radiation. It has been found that 10 microcuries of radioactive sodium per gram of body weight injected subcutaneously *in the normal mouse* are equivalent in effect to 100 roentgens of heavily filtered 200 kv. roentgen rays. *This ratio would not hold for the human; the same number of microcuries would be equivalent to a considerably higher number of roentgens.*

Mice with enlarged nodes and extremely high leukocyte counts were found especially sensitive to the radiosodium. They were not tested with roentgen rays. The marked response was apparently due to radiosensitivity of the abnormal leukocytes rather than to any selective concentration of the material in lymph nodes.*

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* Since the completion of this paper, a 1944 volume of Acta Radiologica has become available; it has been found to contain an article on a similar subject. (Treatment of Leukemia with Artificial Radio-Active Sodium, by Jorgen E. Thygesen, Aage Videboek, and Irgens Villaume. Acta Radiol. 25[1], 305–316, 1944.) In their preliminary experiments with mice, the results were inconclusive. They report animals not able to tolerate more than 1 millicurie in a single dose, which is contrary to our findings. However their experimental procedure differed from ours in several respects,—strain of mouse, type of tumor, method of administration of radio-sodium, and method of standardization of radio-sodium dosage.

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AN ANALYSIS OF THE PHYSICAL FACTORS CONTROLLING THE DIAGNOSTIC QUALITY OF ROENTGEN IMAGES*

PART III. CONTRAST AND THE INTENSITY DISTRIBUTION FUNCTION OF A ROENTGEN IMAGE

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THE clarity with which a roentgen image is reproduced is governed by four principal factors: (*a*) the maximum resolving power of the film or screen on which the image is recorded, (*b*) a relatively minor parameter called the resolution coefficient, (*c*) the contrast exhibited between the image and its surrounding field and (*d*) the unsharpness of the image boundary. In previous articles of this series,[4,5] the relationship which these factors bear to one another has been established and a detailed discussion of maximum resolving power and the resolution coefficient presented. In this and in Part IV attention will be centered on roentgen contrast.

When the term contrast is used to describe the appearance of a roentgenographic or roentgenoscopic image, reference is made to the brightness of the image relative to the brightness of the surrounding field. When the ratio of the two brightness levels is small, contrast is low; when the ratio is large, contrast is high. Visually the relationship between contrast and the brightness ratio of an image is logarithmic. Therefore, it is customary to express contrast quantitatively as

$$C = \log I_i/I_b = \log I_i - \log I_b \qquad (1)$$

where I_i and I_b are the brightness levels or light intensities emitted by the image and by its surrounding field respectively.

I. FACTORS CONTROLLING ROENTGEN CONTRAST

When a roentgen-ray beam is projected through a structure a considerable portion of the radiation is either absorbed or scattered by the various included materials. The relatively small remainder progresses through the structure to emerge unaltered from the other side. The fraction of the radiation which falls in the latter category depends among other things on the atomic composition and densities of the materials transmitting the radiation. Thus, if the structure is composed of bone, muscle and fat tissue, the roentgen-ray intensity of the emergent beam varies from point to point in a plane perpendicular to the direction of the radiation in accordance with the characteristics of the tissues through which the beam passes; that is, the emergent beam presents a series of roentgen-ray images of the various components of the structure. Since the eye does not respond to roentgen radiation, these images may only be perceived when recorded by a roentgenographic film or roentgenoscopic screen.

The brightness levels exhibited by an illuminated film or an irradiated screen are governed by the intensities of the activating roentgen radiation. Contrast, therefore, is primarily determined by the intensity distribution function of the radiation which the film or screen receives and accordingly is a function of the properties of the structure under examination and the characteristics of the roentgen beam.

The relationship between the roentgen-ray intensity transmitted by a homogeneous structure and the structure's thickness and density is illustrated graphically in Fig-

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ure 1 and may be expressed by the equation

$$\log G = \log G_o - \int b\rho dx \qquad (2)$$

where G is the intensity of the roentgen radiation transmitted by the material, G_o is the radiation intensity if no structure were interposed in the roentgen beam, ρ and x are the density and thickness of the material respectively and b is the negative slope of the log G vs. ρx curve.

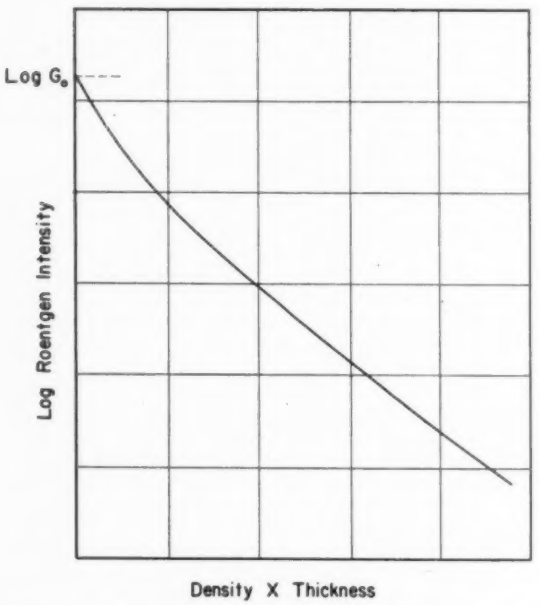

FIG. 1. Relationship between the logarithm of the roentgen-ray intensity transmitted by a structure and the product of the structure's density and thickness.

As pointed out in Part 1 of this series, the factor, b, is the material's mass absorption coefficient expressed in decadic logarithms. Its value is a function of the atomic numbers of the several components comprising the material and of the spectral distribution of the roentgen radiation. It thereby is a function of the voltage applied to the roentgen tube and of any filtration placed in the roentgen beam.

Equation (2) is valid only for a homogeneous structure and then only when scattered radiation is excluded from consideration. When the structure is heterogeneous and scattered radiation is present, the expression requires further development.

The intensity of the primary radiation transmitted by a heterogeneous structure may be related to the absorption coefficients, densities and thicknesses of the several constituents of the structure merely by expanding equation (2); that is,

$$\log G_p = \log G_o - \int_o^{x_1} b_1\rho_1 dx - \int_o^{x_2} b_2\rho_2 dx \cdots - \int_o^{x_n} b_n\rho_n dx \qquad (3)$$

where G_p is the intensity of the primary transmitted radiation and the subscripts, 1, $2 \cdots n$, refer to the absorption coefficients, densities and thicknesses of the n materials included in the structure.

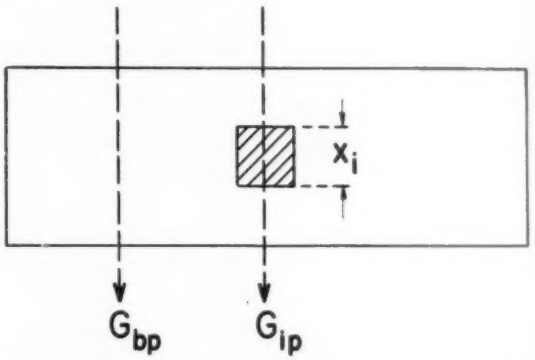

FIG. 2. Schematic diagram illustrating relationships expressed in equations (3a) and (3b).

Now, if a roentgen beam is projected through a heterogeneous structure which includes an image-producing object of thickness, x_i, and density, ρ_i, and having a mass absorption coefficient, b_i, the roentgen-ray intensity of the primary emergent beam directly below the object (see Fig. 2) is given by the equation,

$$\log G_{ip} = \log G_o - \int_o^{x_1} b_1\rho_1 dx - \int_o^{x_2} b_2\rho_2 dx \cdots - \int_o^{x_i} b_i\rho_i dx \cdots - \int_o^{x_n} b_n\rho_n dx. \qquad (3a)$$

Also if this object is surrounded by a material having a density, ρ_b, and a mass absorption coefficient, b_b, the roentgen-ray intensity of the primary emergent beam just outside the object's image is

$$\log G_{bp}=\log G_o-\int_o^{x_1} b_1\rho_1 dx-\int_o^{x_2} b_2\rho_2 dx\cdots -\int_o^{x_i} b_b\rho_b dx\cdots -\int_o^{x_n} b_n\rho_n dx. \quad (3b)$$

When equation (3b) is subtracted from equation (3a)

$$\log G_{ip}-\log G_{bp}=\int_o^{x_i} b_b\rho_b dx-\int_o^{x_i} b_i\rho_i dx. \quad (4)$$

Under most roentgen conditions the thickness of an image-producing object is sufficiently small so that the values of the mass absorption coefficients remain essentially constant over the integral. Under these circumstances then, equation (4) reduces to

$$\log G_{ip}-\log G_{bp}=(b_b\rho_b-b_i\rho_i)x_i. \quad (5)$$

Equation (5) indicates the intensity distribution of the *primary* radiation within a roentgen image and its surrounding field. When scattered radiation is present the intensity distribution function is more complex and is dependent on the size and position of the image-producing object.

Although an object may have a variety of sizes and positions, only two combinations will be discussed at this time: (*a*) the object is small and occupies a position some distance from the roentgenographic film or roentgenoscopic screen; and (*b*) the object is small or large and occupies a position adjacent to the film or screen. As will be shown presently, a knowledge of these conditions will permit an estimation of the intensity distribution functions which occur under most circumstances.

(*a*) *Intensity Distribution Function of Image Produced by Small Object Located a Distance from Film or Screen.* When the image-producing object is small and occupies a position more than a few centimeters from the film or screen, the intensities of the scattered radiation within the image and within the surrounding field are essentially equal (see Fig. 3) because radiation originating in adjoining portions of the structure may freely undercut the object and distribute itself uniformly over the film or screen surface. Furthermore, the scattered radiation arising in the object is dispersed widely inside and outside the image.

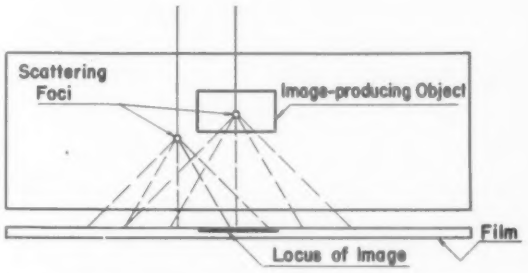

Fig. 3. Schematic diagram illustrating the dispersal of scattered radiation equally inside and outside the boundaries of a roentgen image when the image-producing object occupies a position some distance from the recording film or screen.

If the intensities of the scattered radiation within an image and its surrounding field are equal, the intensities of the total radiation at the respective sites when expressed in logarithmic form will be

$$\log G_{it}=\log (G_{ip}+G_s) \quad (6a)$$

and

$$\log G_{bt}=\log (G_{bp}+G_s) \quad (6b)$$

where G_{it} and G_{bt} are the intensities of the total radiation received within the image and its surrounding field respectively, and G_s is the intensity of the scattered radiation. Therefore, when equation (6b) is subtracted from equation (6a),

$$\log G_{it}-\log G_{bt}=-\log\left[1+\frac{\Delta G}{G_{ip}}\left(\frac{G_{ip}}{G_{ip}+G_s}\right)\right] \quad (7)$$

where

$$\Delta G=(G_{bp}-G_{ip}).$$

Equation (7) may be written in the form of the power series;

$$\log G_{it}-\log G_{bt}=-\frac{1}{2.3}\left[\frac{\Delta G}{G_{ip}}\left(\frac{G_{ip}}{G_{ip}+G_s}\right)-\frac{1}{2}\left(\frac{\Delta G}{G_{ip}}\right)^2\left(\frac{G_{ip}}{G_{ip}+G_s}\right)^2+\cdots\right]. \quad (8)$$

Under almost all roentgen conditions, the terms of this series decrease rapidly with increasing values of the exponents. Therefore, equation (8) may be written

$$\log G_{it}-\log G_{bt}=-\frac{\Delta G}{2.3G_{ip}}\left(\frac{G_{ip}}{G_{ip}+G_s}\right). \quad (8a)$$

Also when the roentgen image is produced by a small object, ΔG is usually considerably less than G_{ip}; therefore

$$-\frac{\Delta G}{2.3G_{ip}}=-\log\left(1+\frac{\Delta G}{G_{ip}}\right)=\log G_{ip}-\log G_{bp} \quad (8b)$$

and

$$\log G_{it}-\log G_{bt}=(\log G_{ip}-\log G_{bp})\left(\frac{G_{ip}}{G_{ip}+G_s}\right). \quad (8c)$$

When equations (5) and (8c) are combined

$$\log G_{it}-\log G_{bt}=(b_b\rho_b-b_i\rho_i)\left(\frac{G_{ip}}{G_{ip}+G_s}\right)x_i. \quad (9)$$

Thus, the presence of scattered radiation reduces the roentgen-ray intensity distribution function of an image by an amount equal to the ratio of the primary radiation intensity to the total radiation intensity.

Equation (9) is valid both when a grid is and is not used. However, it is not in a very convenient form since entirely separate data are required for its solution under the two conditions. This difficulty may be effectively overcome by expanding the function to include the grid efficiency factor.

The efficiency of a grid is determined by its effectiveness in reducing scattered radiation and may be expressed quantitatively by the equation

$$P=\frac{\frac{G_{so}}{G_{po}}-\frac{G_{sg}}{G_{pg}}}{\frac{G_{so}}{G_{po}}} \quad (10)$$

where P is the grid efficiency factor, a parameter which henceforth will be referred to as the *Potter coefficient*,

G_{so} and G_{po} are the intensities of the scattered and primary radiation respectively when a grid is not used and

G_{sg} and G_{pg} are the intensities of the scattered and primary radiation respectively transmitted by the grid.

When equation (10) is rearranged and the factor $(1-P)$ added to each side

$$G_{sg}/G_{pg}+1-P=(1-P)G_{so}/G_{po}+1-P. \quad (10a)$$

Also when equation (10a) is rearranged and inverted

$$\frac{G_{pg}}{G_{pg}+G_{sg}}=\frac{G_{po}/(G_{po}+G_{so})}{1+P[G_{po}/(G_{po}+G_{so})-1]}. \quad (10b)$$

But the left hand term of equation (10b) represents the intensity ratio of primary to total radiation occurring when a grid is employed. Therefore, when equation (10b) is substituted in equation (9), the intensity distribution function becomes

$$\log G_{it}-\log G_{bt}=\frac{(b_b\rho_b-b_i\rho_i)mx_i}{1+P(m-1)} \quad (11)$$

where

$$m=G_{po}/(G_{po}+G_{so}).$$

It will be observed that equation (11) applies equally well to conditions where a grid is not employed if a value of zero is given the Potter coefficient, P, and therefore may be solved with but one set of scattering data regardless of the presence or absence of a grid.

(*b*) *Intensity Distribution Function of Image Produced by Small or Large Object Located in Apposition to the Film or Screen.* When the image-producing object is in close apposition to the film or screen, there is no opportunity for it to be undercut by scattered radiation arising in other portions

of the structure; neither is scattered radiation originating within the object permitted to disperse widely beyond the image boundaries. Accordingly the derivation of the intensity distribution function under this condition follows identically that from which equation (5) was developed with the exception that values of the mass absorption coefficients must be determined from absorption curves made under conditions where scattered radiation is present. The function is simply

$$\log G_{it} - \log G_{bt} = (b_b'\rho_b - b_i'\rho_i)x_i \qquad (12)$$

where the superscripts indicate the special conditions under which the mass absorption coefficients are measured.

As in the case of equation (9), equation (12) is valid both when a grid is and is not used, but is rather inconvenient because entirely separate data are required for its solution under the two conditions. Equation (12), however, is not amenable to simplification by introducing the Potter coefficient if rigorous mathematical analysis is followed. Fortunately, values of b and b' do not differ widely from one another and a close approximation of the value of a material's mass absorption coefficient, when a grid is present, may be obtained from the equation

$$b_g' = (b - b_o')P + b_o' \qquad (12a)$$

where b_g' and b_o' are the mass absorption coefficients under conditions when a grid is and is not used respectively.

When equation (12a) is substituted in equation (12), the intensity distribution function becomes

$$\log G_{it} - \log G_{bt} = \{[b_{bo}' + (b_b - b_{bo}')P]\rho_b - [b_{io}' + (b_i - b_{io}')P]\rho_i\}x_i. \qquad (13)$$

Equation (13) is also valid for conditions when a grid is not used when a value of zero is given the Potter coefficient, P.

Equations (11) and (13) indicate the intensity distribution functions of the radiation within a roentgen image and its surrounding field for two widely different conditions. Since the objects which produce most roentgen images (e.g. a minimal tuberculous process) are relatively small compared to the size of the structure in which they are located and lie some distance from the roentgenographic film or screen, the intensity distribution function usually approximates that given by equation (11). There are, however, a few instances where equation (13) is applicable.

When roentgen radiation impinges on a roentgenoscopic screen the intensity or brightness of the fluorescence is directly proportional to the intensity of the activating radiation; that is,

$$I = kG \qquad (14)$$

where I is the light emission of the screen,
G is the intensity of the roentgen radiation and
k is a constant.

Therefore, if G_{it} and G_{bt} are the roentgen-ray intensities within an image and its surrounding field respectively,

$$\log I_i - \log I_b = \log G_{it} - \log G_{bt}. \qquad (15)$$

But according to equation (1) the left-hand portion of equation (15) is the contrast exhibited by the image; furthermore, the right-hand portion is the intensity distribution function of the roentgen radiation as given by equations (11) and (13). Therefore, the contrast of a roentgenoscopic image is simply equal to the intensity distribution function within the roentgen-ray image and its surrounding field.

If the roentgen radiation is permitted to fall on a roentgenographic film or film-screen combination instead of a roentgenoscopic screen the density of the processed film will be a function of the quantity of radiation (usually referred to as the exposure) received by the film. This relationship, shown graphically in Figure 6, Part 1, may be expressed by the equation

$$D = \int g \, d\log E + K \qquad (16)$$

where g is the slope of the density vs. log exposure curve,
E is the quantity of radiation received by the film and
K is a constant.

The value of the factor, g, is governed by inherent characteristics of the film and the conditions under which the film is developed. Over the small ranges of density which are usually encountered between a roentgenographic image and its surrounding field, it remains essentially constant and for these conditions equation (16) may be written

$$D=g(\log E-i) \tag{16a}$$

where i is a constant.

Now the quantity of radiation received by a film is equal to the product of the effective radiation intensity, G, and the exposure time, t; that is,

$$E=Gt. \tag{17}$$

Therefore, equation (16a) may be written

$$D=g(\log G+\log t-i). \tag{18}$$

Furthermore, if G_{it} and G_{bt} are the intensities within a roentgen-ray image and its surrounding field respectively, the relationship between the resulting roentgenographic densities will be

$$D_i-D_b=g(\log G_{it}-\log G_{bt}) \tag{19}$$

since all portions of the film receive the same exposure time. But by definition the density of a film is a direct function of the logarithm of the light intensity transmitted by the film. Accordingly, the left side of equation (19) represents the contrast exhibited by the image; that is,

$$C_f=g(\log G_{it}-\log G_{bt}). \tag{20}$$

It is evident from equation (20) that the contrast of a roentgenographic image is simply equa to the product of g, a factor which may be conveniently called the film contrast factor, and the intensity distribution function of the roentgen radiation as given by equations (11) and (13). Indeed equation (20) is valid for both roentgenographic and roentgenoscopic images if a value of unity is given the factor, g, in the latter instance.

An examination of equations (11) and (13) reveals that the contrast of a roentgen image is governed by a complex series of factors, including the thickness and density of the image-producing object. Also, since the mass absorption coefficient of a material is a function of the spectral distribution (quality) of the exposing radiation and of the atomic composition of the material, it is affected by the voltage applied to the roentgen tube, by filtration placed in the roentgen beam and by the absorption characteristics of the image-producing object. Finally, since the intensity ratio of primary to total radiation within an image is also a function of the size of the portal irradiated, and the efficiency of a grid if used, these factors also exert control over contrast.

II. Measurement of Factors Controlling Roentgen Contrast

The quantitative manner in which these factors control the contrast of a roentgen image may be evaluated from a study of data which are obtained when pertinent values are substituted in the terms of equations (11) and (13). Such values, however, are not generally available because until recently the measurement of the mass absorption coefficients of bone, muscle, fat and other tissues and of the intensity ratios of primary to total radiation occurring under various roentgen conditions has been an extremely tedious task.

Since the roentgen-ray absorption of a material is a function of the quality of the exposing radiation and since the quality of radiation encountered under roentgenographic and roentgenoscopic conditions extends through a wide range, it is essential that the instruments with which the absorption data of a material are obtained have essentially identical roentgen-ray spectral responses to those of roentgenoscopic screens and roentgenographic films exposed either with or without intensifying screens. Otherwise the results will not reflect the behavior of these screens and films when recording a roentgen image. In the past, two methods have been used to measure the roentgen-ray absorption of a material: one is the ionometric method, widely

used in roentgen therapy, in which the measurements are recorded with an ionization chamber-electrometer system; the second is the film sensitometric method in which roentgenographic film is employed as the radiation recording medium. The former is a rather simple procedure but unfortunately the spectral responses of available ionization chambers do not correspond to those of films and screens and therefore does not yield reliable information. The latter method is completely satisfactory from the standpoint of the spectral response of the recording agent if certain precautions are taken, but it presents serious difficulties when a considerable quantity of data is to be gathered because of the complexity of the procedure. For each set of conditions for which data are desired, several roentgenographic exposures must be made. The films subsequently require processing under closely identical conditions, their densities read and the results analyzed and converted to intensitometric values. It is evident that the film sensitometric method is an impractical procedure when data are to be collected through the wide range of conditions normally encountered in roentgen practice and it is not surprising that values of the absorption coefficients of various anatomical tissues and of the intensity ratios of primary to total radiation which occur under the many roentgen conditions to be expected have not been determined.

The recent development of a photoelectric roentgen-ray intensitometer[3] whose spectral response may be made to coincide with that of any roentgenographic or roentgenoscopic material and with which measurements may be made quickly and easily has greatly simplified the collection of such data, however. With this instrument it has been possible to obtain much quantitative information regarding the factors which control roentgen contrast.

The measurement of the mass absorption coefficients of bone, muscle and fat cannot be conveniently conducted with living tissues and it is therefore necessary to employ substitute phantom materials whose absorption characteristics are closely similar. This problem, however, presents no serious difficulty. The mass absorption coefficient of a material is governed by the material's atomic composition and is independent of its molecular structure. Furthermore, the atomic compositions of tissues other than bone and pathological calcifications are rather similar and these structures therefore exhibit reasonably identical absorption characteristics. Accordingly, only two phantom materials, one to represent calcified and one to represent non-calcified tissues are required, and these materials, if desired, need not have chemical properties even remotely similar to the tissues for which they substitute.

Masonite presdwood, a cellulose building material having a density of 1.07, exhibits absorption characteristics similar to fat, muscle and connective tissue and, as will be pointed out presently, constitutes an excellent phantom material to represent these structures. A suitable phantom material to represent the inorganic constituents of bone and of other calcifications is also readily obtainable. It has been shown[2] that these constituents occur as carbonate apatite having the formula $Ca_{10}CO_3(PO_4)_6 \cdot H_2O$. Accordingly a powdered mixture of calcium phosphate and calcium carbonate in proportions by weight of 9.3 to 1.0 will possess comparable absorption characteristics.

Absorption measurements have been made with both of these phantoms through a wide range of conditions in the laboratories of The University of Chicago and the United States Public Health Service. In the case of Masonite presdwood, a phantom 30 cm. square and ranging in thickness from 0 to 30 cm. was employed. Data were obtained with the roentgen machine, a four-valve, full-wave, single-phase unit operating at 30, 50, 70 and 85 kv. (peak), and with an inherent tube filtration of 0.5 mm. Al. The measurements were repeated with an added filter of 5.0 mm. Al. The sizes of the portal irradiated were 300 sq. cm. and 900 sq. cm. measured at the level

of the photoelectric intensitometer; in addition, a set of data was obtained under conditions when essentially no scattered radiation was permitted to reach the intensitometer. Under each circumstance for which an absorption measurement was

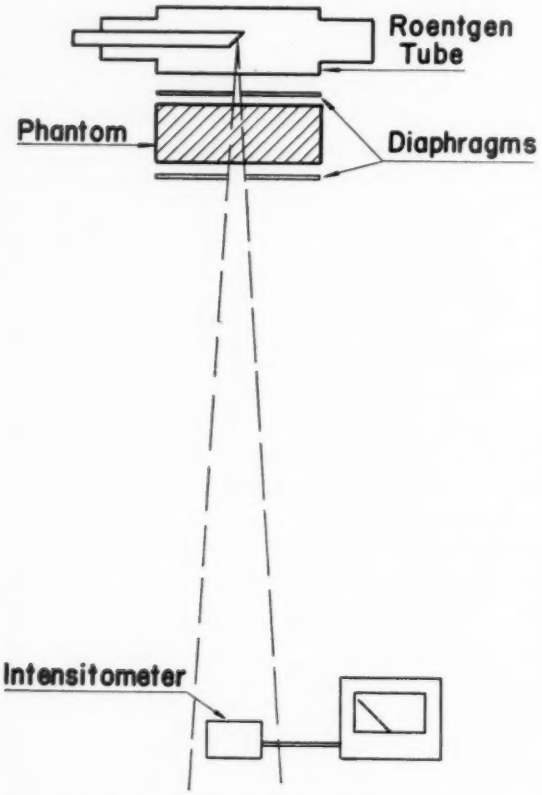

FIG. 4. Schematic diagram illustrating the arrangement by which absorption measurements were made in the absence of scattered radiation. The lead diaphragms limit the size of the roentgen beam to levels at which the quantity of scattered radiation is a minimum, and the large phantom-intensitometer distance attenuates the small quantity of scattered radiation that is produced to insignificantly low levels (less than 1 per cent of the intensity of the primary radiation).

made, readings were taken both when the intensitometer was adjusted to have a spectral response similar to that of roentgenographic films exposed with intensifying screens and when adjusted to have a response identical to that of films exposed directly to roentgen radiation. Films exposed with intensifying screens have absorption characteristics similar to those of photofluorographic films exposed with a Patterson type B screen and to those of Patterson type B roentgenoscopic screens.[3] The data obtained with the intensitometer having a film-screen spectral response are therefore also applicable to these conditions. Furthermore, films exposed with roentgen rays directly have a spectral response identical to that of photofluorographic films exposed with the Patterson type D screen (photoroentgen); the non-screen data are therefore applicable to this condition.

The absorption measurements in which scattered radiation was prevented from falling on the intensitometer were made under conditions illustrated in Figure 4. It will be observed that the phantom was located at a position close to the roentgen tube whereas the radiation detector of the photoelectric intensitometer was placed 2 meters below. In addition, the roentgen beam was carefully diaphragmed above and below the phantom to reduce the production and dispersal of scattered radiation. Intensitometric measurements with the phantom located at various positions between the radiation detector and the roentgen tube indicated that the intensity of the scattered radiation when the phantom was located in the position shown in Figure 4 was less than 1 per cent of that of the primary radiation. This method of effectively eliminating scattered radiation was first suggested by Seemann.[6]

The absorption data for various thicknesses of presdwood, irradiated at roentgen tube potentials of 30, 50, 70 and 85 kv. (peak) through portals of 0.0, 300 and 900 sq. cm., with a roentgen beam filtration of 0.5 mm. Al (inherent tube filtration) and with the photoelectric intensitometer adjusted to have a spectral response similar to that of films exposed with intensifying screens are illustrated graphically in Figure 5. Similar data, obtained with a roentgen beam filtration of 5.5 mm. Al are presented in Figure 6. In Figures 7 and 8 the data apply to conditions where films are exposed to roentgen rays directly; in the former

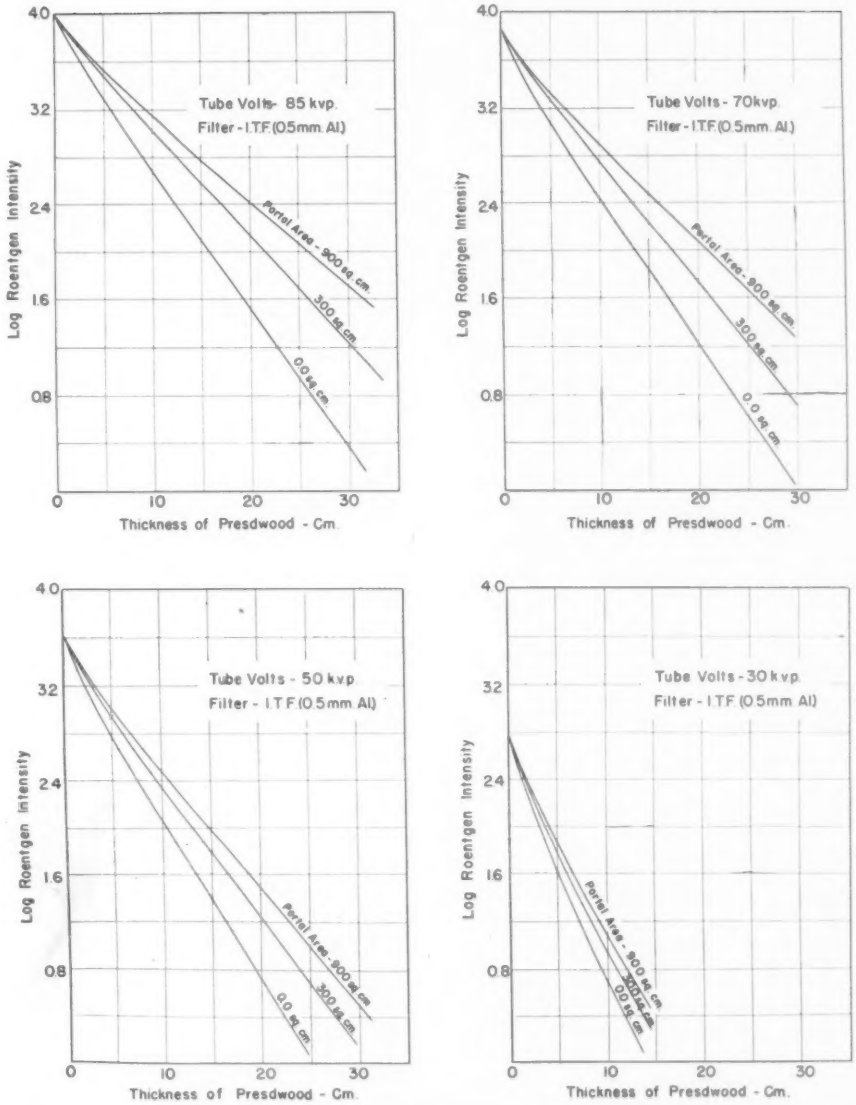
Tube Volts- 85 kvp.
Filter - I.T.F.(0.5mm. Al.)
Portal Area - 900 sq. cm.
300 sq. cm.
0.0 sq. cm.
Log Roentgen Intensity
Thickness of Presdwood - Cm.
Tube Volts - 70kvp.
Filter - I.T.F.(0.5mm. Al.)
Tube Volts - 50 kvp.
Filter - I.T.F.(0.5mm. Al.)
Tube Volts - 30 kvp.
Filter - I.T.F.(0.5mm. Al.)

FIG. 5. Absorption curves for Masonite presdwood (density = 1.07) irradiated with a four-valve, full-wave rectified roentgen generator operating at potentials of 30, 50, 70, and 85 kv. (peak), through portals of 0.0, 300 and 900 sq. cm., with a roentgen-ray beam filtration of 0.5 mm. Al (inherent tube filtration) and with the photoelectric intensitometer adjusted to have a spectral response similar to that of films exposed with intensifying screens.

case, readings were made with an inherent tube filtration of 0.5 mm. Al, and in the latter, with a total filtration of 5.5 mm. Al. The portal size of 0.0 sq. cm. refers to the condition where scattered radiation was prevented from reaching the intensitometer. Since in practice an absence of scattered radiation can occur only when the portal size is zero, this method of designation has been adopted.

The values of the mass absorption coefficient of Masonite presdwood under conditions when scattered radiation is prevented from reaching the film or screen (i.e. values which are applicable to equation (11)) may be determined by simply measuring the slopes of the 0.0 sq. cm. curves of Figures 5 to 8. These values are tabulated in Tables I to IV. In Table I, the data are presented for various thicknesses of presdwood when

TABLES I–IV

MASS ABSORPTION COEFFICIENTS OF MASONITE PRESDWOOD EXPRESSED IN TERMS OF DECADIC LOGARITHMS
THESE VALUES ARE CLOSELY EQUIVALENT TO THOSE OF ALL ANATOMIC MATERIALS WITH
THE EXCEPTION OF CALCIFIED TISSUES

TABLE I

Data applicable to conditions where:
Exposures made with intensifying screens.
Filtration in roentgen beam = Inherent tube filtration (0.5 mm. Al)

Thickness of Presdwood (cm.)	Kilovolts (peak)			
	85	70	50	30
0.0	0.210	0.231	0.262	0.300
2.5	0.129	0.143	0.163	0.205
5.0	0.117	0.125	0.136	0.173
7.5	0.110	0.114	0.125	0.165
10.0	0.109	0.113	0.124	0.164
12.5	0.108	0.112	0.123	0.164
15.0	0.107	0.111	0.122	
17.5	0.107	0.110	0.121	
20.0	0.106	0.109	0.120	
22.5	0.106	0.109	0.119	
25.0	0.105	0.108	0.118	
27.5	0.105	0.108	0.117	
30.0	0.104	0.107	0.117	

TABLE II

Data applicable to conditions where:
Exposures made with intensifying screens.
Filtration in roentgen beam = Inherent tube filtration (0.5 mm. Al) plus 5.0 mm. Al

Thickness of Presdwood (cm.)	Kilovolts (peak)		
	85	70	50
0.0	0.107	0.110	0.125
2.5	0.105	0.109	0.120
5.0	0.104	0.108	0.118
7.5	0.104	0.108	0.117
10.0	0.103	0.107	0.117
12.5	0.103	0.107	0.117
15.0	0.103	0.107	0.117
17.5	0.103	0.107	0.117
20.0	0.103	0.107	0.117
22.5	0.103	0.107	0.117
25.0	0.103	0.107	0.117
27.5	0.103	0.107	0.117
30.0	0.103	0.107	0.117

TABLE III

Data applicable to conditions where:
Exposures made without intensifying screens (cardboard holders).
Filtration in roentgen beam = Inherent tube filtration (0.5 mm. Al)

Thickness of Presdwood (cm.)	Kilovolts (peak)			
	85	70	50	30
0.0	0.235	0.255	0.283	0.330
2.5	0.142	0.155	0.175	0.220
5.0	0.125	0.133	0.145	0.180
7.5	0.117	0.121	0.130	0.168
10.0	0.113	0.117	0.127	
12.5	0.110	0.115	0.125	
15.0	0.108	0.112	0.123	
17.5	0.107	0.110	0.122	
20.0	0.106	0.110	0.121	

TABLE IV

Data applicable to conditions where:
Exposures made without intensifying screens (cardboard holders).
Filtration in roentgen beam = Inherent tube filtration (0.5 mm. Al) plus 5.0 mm. Al

Thickness of Presdwood (cm.)	Kilovolts (peak)		
	85	70	50
0.0	0.110	0.120	0.132
2.5	0.106	0.113	0.125
5.0	0.104	0.111	0.120
7.5	0.103	0.108	0.118
10.0	0.103	0.107	0.117
12.5	0.103	0.107	0.117
15.0	0.103	0.107	0.117
17.5	0.103	0.107	0.117
20.0	0.103	0.107	0.117

irradiated at the four kilovoltages studied, under conditions where the radiation is received by films exposed with intensifying screens. Similar data for conditions where the roentgen beam filtration is 5.5 mm. Al are listed in Table II. Tables III and IV are for conditions where the radiation is received by films exposed without intensifying screens; the former applies to a roentgen beam filtration of 0.5 mm. Al and the latter to a filtration of 5.5 mm. Al.

The values of the mass absorption coeffi-

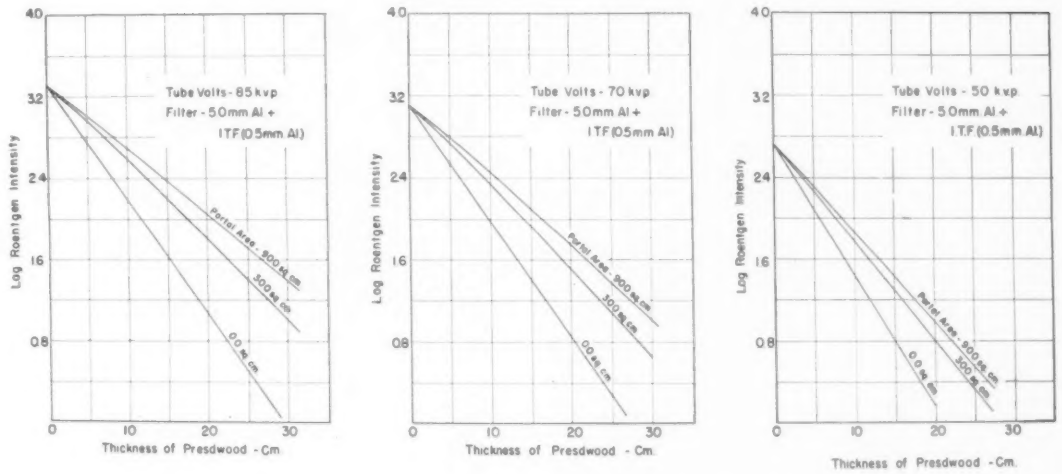
Tube Volts - 85 kvp
Filter - 50mm Al +
ITF (0.5mm Al)
Portal Area - 900 sq. cm.
300 sq. cm.
0.0 sq. cm.
Log Roentgen Intensity
Thickness of Presdwood - Cm.
Tube Volts - 70 kvp
Filter - 50mm Al +
ITF (0.5mm Al)
Portal Area - 900 sq. cm.
300 sq. cm.
0.0 sq. cm.
Log Roentgen Intensity
Thickness of Presdwood - Cm.
Tube Volts - 50 kvp
Filter - 50mm Al +
ITF (0.5mm Al)
Portal Area - 900 sq. cm.
300 sq. cm.
0.0 sq. cm.
Log Roentgen Intensity
Thickness of Presdwood - Cm.

FIG. 6. Absorption curves for Masonite presdwood (density = 1.07) irradiated with a four-valve, full-wave rectified roentgen generator operating at potentials of 50, 70 and 85 kv. (peak), through portals of 0.0, 300 and 900 sq. cm., with a roentgen-ray beam filtration of 5.5 mm. Al and with the photoelectric intensitometer adjusted to have a spectral response similar to that of films exposed with intensifying screens.

cients for presdwood listed in Tables I to IV are not, of course, exactly representative of those of bone, muscle, fat and other tissue. They are, however, closely similar as will be evident from Figure 9 where absorption curves for fresh skeletal muscle (beef), presdwood, and lard (animal fat), are plotted for roentgen tube potentials of 30, 50, 70 and 85 kv. (peak). These measurements were obtained under conditions where the intensity of the scattered radiation reaching the intensitometer was effectively zero and where the intensitometer's spectral response was similar to that of films exposed with intensifying screens. The comparative values of the absorption coefficients of the three materials at the four kilovoltages are listed in Table V, a value of 100 per cent being assigned to Masonite presdwood in each case.

The data provided in Figures 5 to 8 may also be used to calculate the intensity ratios of primary to total radiation which occur under roentgenographic and roentgenoscopic conditions for portal areas of 300 and 900 sq. cm. The values for conditions where films are exposed with intensifying screens are listed in Tables VI to IX and for conditions where films are exposed to roentgen rays directly in Tables X to XIII. In each case the figures apply respectively to (1) a portal area of 300 sq. cm. and a roentgen beam filtration of 0.5 mm. Al (inherent), (2) a portal area of 300 sq. cm. and a total filtration of 5.5 mm. Al, (3) a portal area of 900 sq. cm., and 0.5 mm. Al filtration (inherent) and (4) a portal area of 900 sq. cm. and 5.5 mm. Al filtration (total).

TABLE V

COMPARATIVE VALUES OF THE MASS ABSORPTION COEFFICIENTS OF ANIMAL FAT, PRESDWOOD AND SKELETAL MUSCLE

Material	Kilovoltage (peak)			
	30	50	70	85
Animal fat	0.92	0.94	0.96	0.97
Masonite presdwood	1.00	1.00	1.00	1.00
Skeletal muscle	1.04	1.03	1.02	1.02

Absorption curves for $Ca_{10}CO_3(PO_4)_6$, (density = 1.00) irradiated at roentgen tube potentials of 30, 50, 70 and 85 kv. (peak), with a roentgen beam filtration of 0.5 mm. Al (inherent tube filtration) with the photoelectric intensitometer adjusted to have a spectral response similar to that of films exposed with intensifying

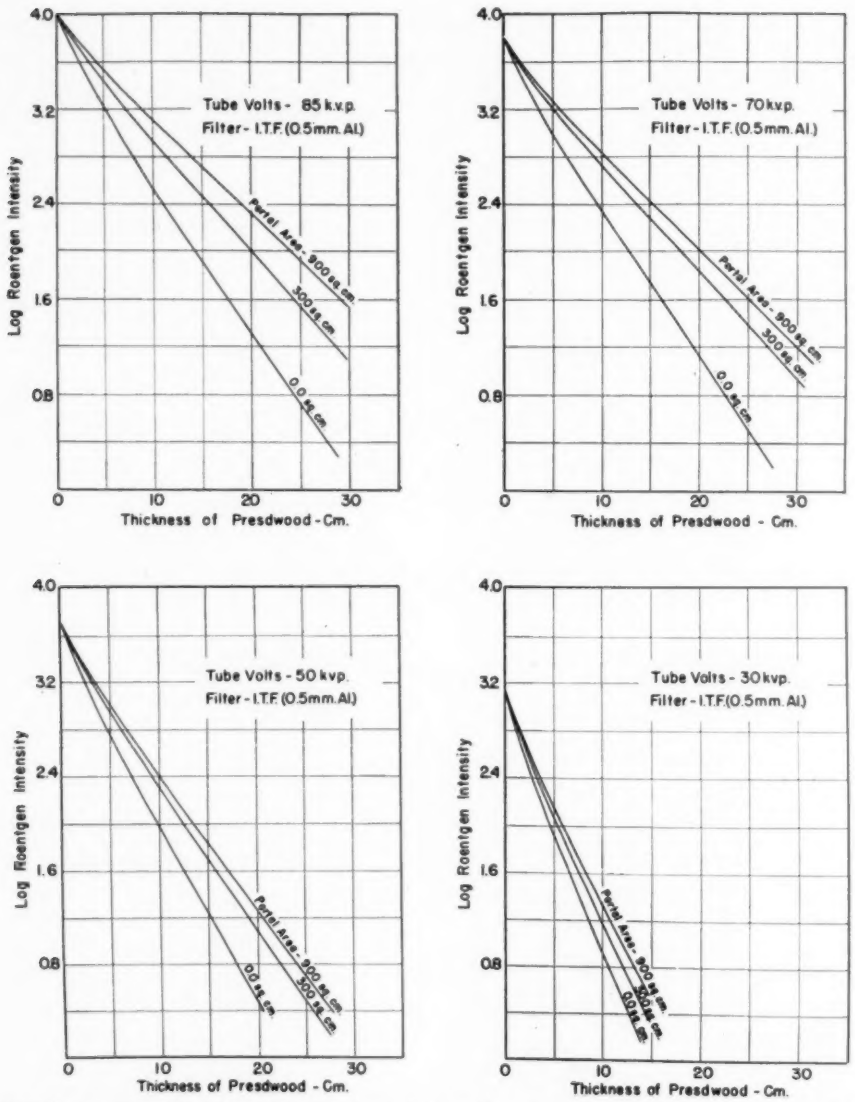
Tube Volts - 85 kvp.
Filter - I.T.F. (0.5mm. Al)
Tube Volts - 70 kvp.
Filter - I.T.F. (0.5mm. Al)
Tube Volts - 50 kvp.
Filter - I.T.F. (0.5mm. Al)
Tube Volts - 30 kvp.
Filter - I.T.F. (0.5mm. Al)
Log Roentgen Intensity
Thickness of Presdwood - Cm.
Portal Area - 900 sq. cm.
300 sq. cm.
0.0 sq. cm.

FIG. 7. Absorption curves for Masonite presdwood (density = 1.07) irradiated with a four-valve, full-wave rectified generator operating at potentials of 30, 50, 70, and 85 kv. (peak), through portals of 0.0, 300 and 900 sq. cm., with a roentgen-ray beam filtration of 0.5 mm. Al (inherent tube filtration) and with the photoelectric intensitometer adjusted to have a spectral response similar to that of films exposed to roentgen rays directly.

screens and with scattered radiation reaching the intensitometer effectively reduced to zero are illustrated in Figure 10. Curves *A* to *F* refer respectively to conditions where additional filters of Masonite presdwood (density 1.07) 0.0 cm., 2.5 cm., 5.0 cm., 10 cm., 20 cm., and 30 cm. in thickness are present. Similar data obtained with a roentgen beam filtration of 5.5 mm. Al are shown in Figure 11. In Figures 12 and 13 the data apply to conditions where films are exposed to roentgen rays directly; in the former case, readings were made with an inherent tube filtration of 0.5 mm. Al, and in the latter with a total filtration (exclusive of presdwood) of 5.5 mm. Al.

In Tables XIV to XXXIII are listed the values of the mass absorption coefficient of $Ca_{10}CO_3(PO_4)_6$ which occur under the foregoing conditions. They were determined simply by measuring the slopes of the curves in Figures 10 to 13.

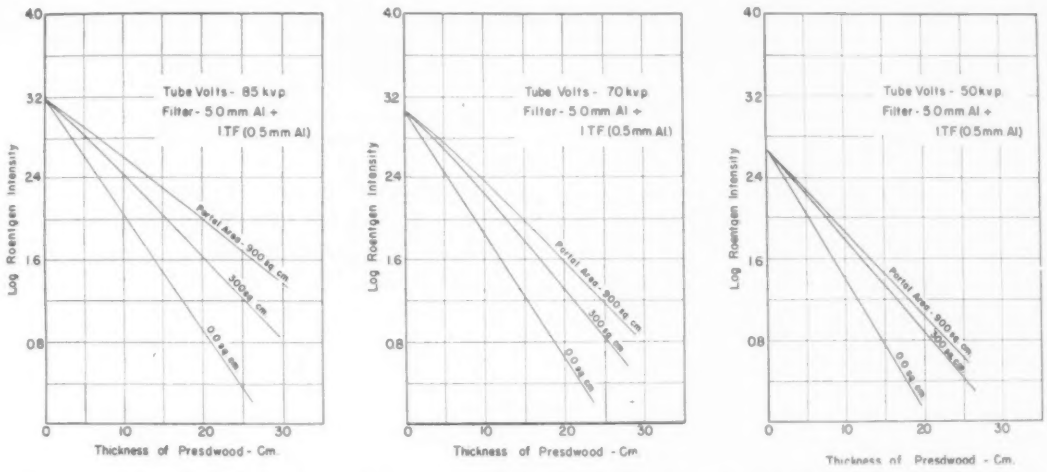
Tube Volts - 85 kvp
Filter - 5.0 mm Al +
1 T F (0.5 mm Al)
Portal Area - 900 sq. cm.
300 sq. cm.
0.0 sq. cm.
Log Roentgen Intensity
Thickness of Presdwood - Cm.
Tube Volts - 70 kvp
Tube Volts - 50 kvp

FIG. 8. Absorption curves for Masonite presdwood (density = 1.07) irradiated with a four-valve, full-wave rectified generator operating at potentials of 50, 70, and 85 kv. (peak), through portals of 0.0, 300 and 900 sq. cm., with a total roentgen beam filtration of 5.5 mm. Al and with the photoelectric intensitometer adjusted to have a spectral response similar to that of films exposed to roentgen rays directly.

DISCUSSION

It was shown in equation (20) that the contrast of a roentgenographic or roentgenoscopic image is proportional to the image's roentgen-ray intensity distribution function usually given by equation (11) or (13). It is therefore possible with the data which have just been presented to evaluate the manner in which contrast is affected by various roentgen conditions. Thus if F_1 and F_2 are the intensity distribution functions for two different circumstances, the percentage change in contrast which occurs when one progresses from one condition to the other will be

$$\%\Delta C = \frac{F_2 - F_1}{F_1} \times 100. \qquad (21)$$

In the following paragraphs several illustrative examples are given to demonstrate the manner in which equation (21) may be employed.

(*a*) *Effect of Kilovoltage on Roentgen Contrast.* A perusal of the tables indicates that the changes in the mass absorption coefficients which occur when the kilovoltage applied to the roentgen tube is altered are

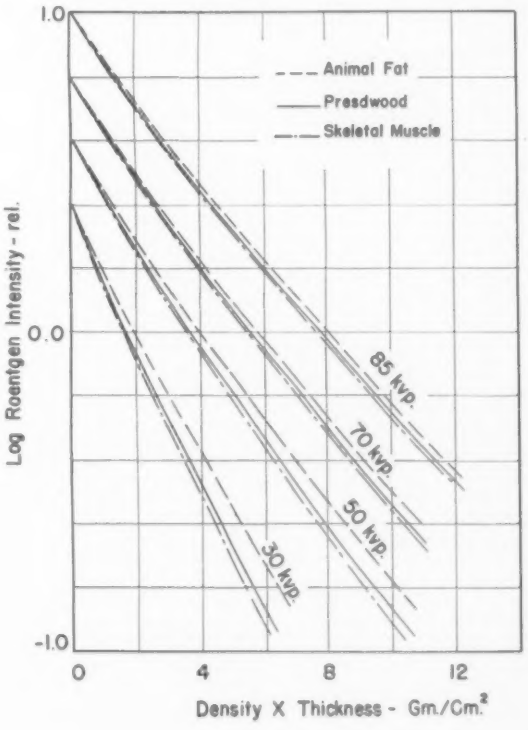

FIG. 9. Absorption curves for animal fat (density = 0.92), Masonite presdwood (density = 1.07) and skeletal beef muscle (density = 1.05), irradiated with a four-valve, full-wave rectified generator operating at potentials of 30, 50, 70, and 85 kv. (peak), with a roentgen-ray beam filtration of 0.5 mm. Al, and with the photoelectric intensitometer adjusted to have a spectral response similar to that of films exposed with intensifying screens. These data were obtained in the absence of scattered radiation.

Tables VI to IX

RATIOS OF PRIMARY TO TOTAL RADIATION RECEIVED BY ROENTGENOGRAPHIC FILMS EXPOSED WITH INTENSIFYING SCREENS AND BY ROENTGENOSCOPIC SCREENS (PATTERSON TYPE "B"). DATA INCLUDE VALUES FOR PHANTOM THICKNESSES RANGING FROM 0.0. TO 30 CM. OF MASONITE PRESDWOOD (DENSITY = 1.07 CM.), FOR FOUR KILOVOLTAGES, FOR TWO PORTAL AREAS AND FOR TWO DEGREES OF FILTRATION

Table VI

Data applicable to conditions where:

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al)

Portal area = 300 sq. cm.

Thickness of Presdwood (cm.)	Kilovolts (peak)			
	85	70	50	30
0.0	1.000	1.000	1.000	1.000
2.5	0.740	0.755	0.770	0.805
5.0	0.620	0.635	0.650	0.690
7.5	0.530	0.550	0.565	0.615
10.0	0.455	0.475	0.500	0.560
12.5	0.385	0.415	0.445	
15.0	0.330	0.360	0.395	
17.5	0.280	0.310	0.350	
20.0	0.240	0.270	0.310	
22.5	0.205	0.235		
25.0	0.175	0.205		
27.5	0.150			
30.0	0.130			

Table VII

Data applicable to conditions where:

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al)

Portal area = 900 sq. cm.

Thickness of Presdwood (cm.)	Kilovolts (peak)			
	85	70	50	30
0.0	1.000	1.000	1.000	1.000
2.5	0.700	0.715	0.730	0.745
5.0	0.540	0.555	0.570	0.590
7.5	0.420	0.445	0.460	0.480
10.0	0.325	0.350	0.380	0.410
12.5	0.250	0.275	0.310	
15.0	0.195	0.220	0.255	
17.5	0.150	0.175	0.215	
20.0	0.115	0.135	0.180	
22.5	0.090	0.110		
25.0	0.070	0.087		
27.5	0.0540			
30.0	0.0415			

Table VIII

Data applicable to conditions where:

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al) plus 5.0 mm. Al

Portal area = 300 sq. cm.

Thickness of Presdwood (cm.)	Kilovolts (peak)		
	85	70	50
0.0	1.000	1.000	1.000
2.5	0.760	0.750	0.790
5.0	0.630	0.620	0.655
7.5	0.525	0.525	0.550
10.0	0.440	0.440	0.470
12.5	0.370	0.375	0.400
15.0	0.310	0.320	0.340
17.5	0.260	0.270	0.295
20.0	0.215	0.230	0.250
22.5	0.180	0.195	
25.0	0.155	0.165	
27.5	0.130		
30.0	0.105		

Table IX

Data applicable to conditions where:

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al) plus 5.0 mm. Al.

Portal area = 900 sq. cm.

Thickness of Presdwood (cm.)	Kilovolts (peak)		
	85	70	50
0.0	1.000	1.000	1.000
2.5	0.750	0.710	0.750
5.0	0.560	0.535	0.570
7.5	0.420	0.415	0.455
10.0	0.325	0.325	0.365
12.5	0.245	0.255	0.290
15.0	0.185	0.195	0.230
17.5	0.140	0.150	0.180
20.0	0.105	0.120	0.145
22.5	0.082	0.093	
25.0	0.062	0.072	
27.5	0.047		
30.0	0.037		

much greater for calcified than for non-calcified tissues. It will therefore be necessary to treat the problem in two parts.

First, let us consider an image produced by a plane of fat tissue, having a density of 0.92, which separates two muscle bundles,

Tables X to XIII

RATIOS OF PRIMARY TO TOTAL RADIATION RECEIVED BY ROENTGENOGRAPHIC FILMS EXPOSED WITHOUT INTENSIFYING SCREENS. DATA INCLUDE VALUES FOR RANGE OF PHANTOM THICKNESS EXTENDING FROM 0.0 TO 20 CM. OF MASONITE PRESDWOOD (DENSITY = 1.07 CM.), FOR FOUR KILOVOLTAGES, FOR TWO PORTAL AREAS AND FOR TWO DEGREES OF FILTRATION

Table X

Data applicable to conditions where:
Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al)
Portal area = 300 sq. cm.

Thickness of Presdwood (cm.)	Kilovolts (peak)			
	85	70	50	30
0.0	1.000	1.000	1.000	1.000
2.5	0.710	0.725	0.740	0.780
5.0	0.565	0.590	0.605	0.655
7.5	0.465	0.490	0.515	0.565
10.0	0.390	0.420	0.450	0.500
12.5	0.330	0.360	0.390	
15.0	0.285	0.310	0.345	
17.5	0.240	0.265	0.305	
20.0	0.205	0.230	0.270	

Table XI

Data applicable to conditions where:
Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al)
Portal area = 900 sq. cm.

Thickness of Presdwood (cm.)	Kilovolts (peak)			
	85	70	50	30
0.0	1.000	1.000	1.000	1.000
2.5	0.675	0.690	0.700	0.740
5.0	0.480	0.500	0.525	0.580
7.5	0.365	0.390	0.425	0.480
10.0	0.275	0.310	0.350	0.400
12.5	0.210	0.245	0.290	
15.0	0.165	0.195	0.240	
17.5	0.130	0.155	0.200	
20.0	0.100	0.125	0.165	

Table XII

Data applicable to conditions where:
Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al) plus 5.0 mm. Al
Portal area = 300 sq. cm.

Thickness of Presdwood (cm.)	Kilovolts (peak)		
	85	70	50
0.0	1.000	1.000	1.000
2.5	0.740	0.740	0.740
5.0	0.600	0.590	0.585
7.5	0.490	0.475	0.490
10.0	0.405	0.395	0.415
12.5	0.335	0.330	0.350
15.0	0.280	0.280	0.300
17.5	0.235	0.240	0.260
20.0	0.195	0.205	0.225

Table XIII

Data applicable to conditions where:
Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al) plus 5.0 mm. Al
Portal area = 900 sq. cm.

Thickness of Presdwood (cm.)	Kilovolts (peak)		
	85	70	50
0.0	1.000	1.000	1.000
2.5	0.700	0.700	0.715
5.0	0.500	0.500	0.540
7.5	0.380	0.370	0.415
10.0	0.280	0.280	0.330
12.5	0.210	0.220	0.260
15.0	0.160	0.165	0.210
17.5	0.120	0.135	0.170
20.0	0.092	0.110	0.145

each having a density of 1.05 and a thickness of 1 cm. Let us further imagine that these tissues are located in a structure 7.5 cm. in thickness (e.g. the arm) and occupy a position several centimeters from a film which is to be exposed without intensifying screens. If the roentgen beam filtration is 0.5 mm. Al, the area to be irradiated is 300 sq. cm. and a grid is not used, it is evident from Tables III, V and X that the intensity distribution function (equation (11)) at 70 kv. (peak) is

$$F_1 = (0.121 \times 1.02 \times 1.05 - 0.121 \times 0.96 \times 0.92)(0.490 \times 1.0) = 0.0113$$

and at 50 kv. (peak) is

$$F_2 = (0.130 \times 1.03 \times 1.05 - 0.130 \times 0.94 \times 0.92)(0.515 \times 1.0) = 0.0142.$$

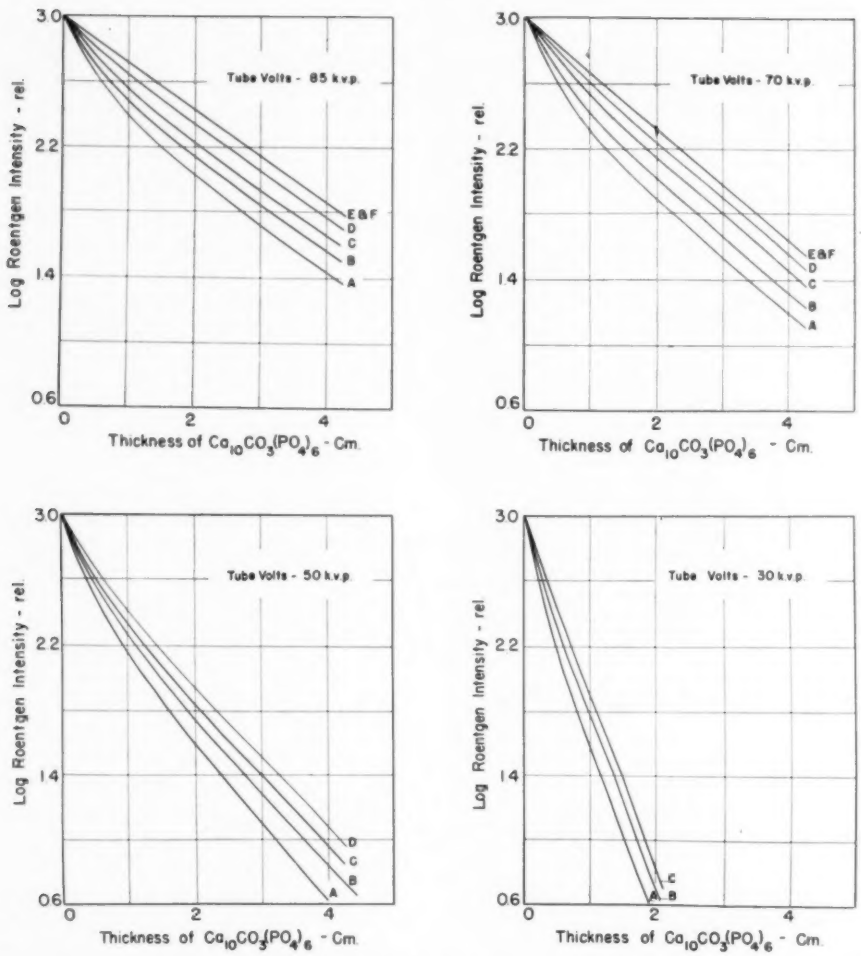

FIG. 10. Absorption curves for carbonate apatite (density = 1.00) irradiated with a four-valve, full-wave rectified generator operating at potentials of 30, 50, 70 and 85 kv. (peak), with a roentgen-ray beam filtration of 0.5 mm. Al (inherent tube filtration), with a photoelectric intensitometer adjusted to have a spectral response similar to that of films exposed with intensifying screens and with scattered radiation reaching the intensitometer effectively reduced to zero. Curves *A* to *F* refer respectively to conditions where additional filtration of 0.0 cm., 2.5 cm., 5.0 cm., 10 cm., 20 cm., and 30 cm. of Masonite presdwood (density = 1.07) is present.

Therefore, from equation (21) the contrast of the image increases approximately 25 per cent between the two conditions. It may be easily shown that this change is somewhat smaller when a grid is used and when the thickness of the structure is greater than 7.5 cm.

A change in contrast of 25 per cent is relatively small and is just beyond the lower limit of perciptibility. The effect of kilovoltage on soft tissue contrast is therefore not great.

If the image were produced by a calcified structure having a density of 2.0 and a thickness of 0.25 cm. and surrounded by skeletal muscle, the intensity distribution functions at 70 and 50 kv. (peak) would be (see Tables III, V, IX, XXVIII and XXIX)

$$F_1 = (0.121 \times 1.02 \times 1.05 - 0.55 \times 2.0)(0.490 \times 0.5) = 0.117$$

and

$$F_2 = (0.130 \times 1.03 \times 1.05 - 0.70 \times 2.0)(0.515 \times 0.5) = 0.163.$$

Such a decrease in kilovoltage would therefore increase the contrast of the image by 40 per cent, an amount somewhat above the minimum level of perceptibility. In this derivation interpolated values of the mass absorption coefficient of $Ca_{10}CO_3(PO_4)_6$ were used.

It may seem strange to some readers that the influence of kilovoltage on contrast is not greater than that indicated in the examples just cited for there are several reports in the roentgenological literature which lead one to expect a much larger effect. These views, however, are based on the belief that the value of the mass absorption coefficient of a material is a function of the third power of the wavelength of the exposing radiation and therefore of the third power of the reciprocal of the potential impressed on the roentgen tube. Abundant experimental evidence does not support the existence of such a simple relationship. Indeed, the mass absorption coefficient of a material appears to be composed of two parts, a photoelectric coefficient and a scattering coefficient, which are related according to the equation

$$2.3\rho b = \tau + \sigma \qquad (22)$$

where b is the mass absorption coefficient expressed in terms of decadic logarithms and τ and σ are the photoelectric and scattering coefficients respectively expressed in terms of Naperian logarithms. The value of the photoelectric coefficient is a function of the third power of the exposing radiation and of the fourth power of the material's atomic number whereas that of the scattering coefficient is relatively independent of both of these parameters.* Within the range of potential from 40 to 85 kv. (peak), the values of the photoelectric coefficient of a material composed of elements of low atomic number such as hydrogen, carbon, nitrogen and oxygen, are small compared to those of the material's scattering coefficient. Accordingly, the values of the mass absorption coefficients of muscle, fat and other non-calcified tissues are largely unaffected by the wavelength of the exposing radiation under these conditions, and changes in roentgen tube kilovoltage therefore do not have a profound influence on contrast.

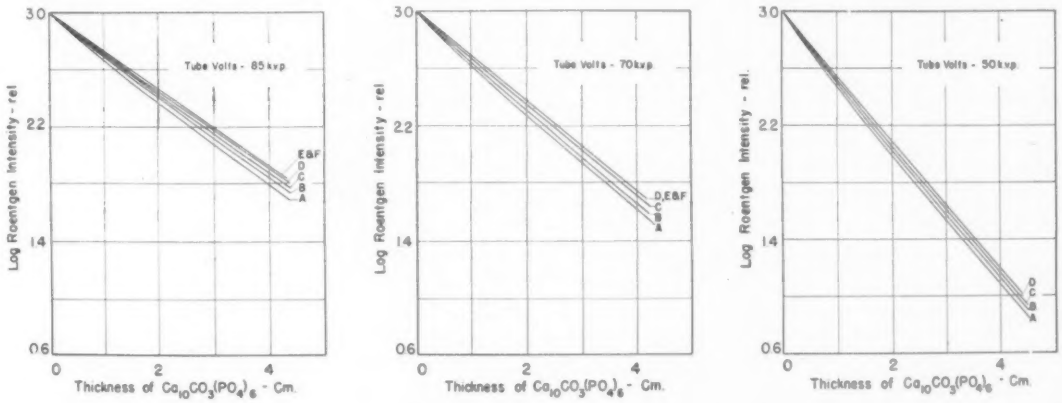

FIG. 11. Absorption curves of carbonate apatite irradiated with a four-valve, full-wave rectified generator operating at potentials of 50, 70 and 85 kv. (peak), with a total roentgen-ray beam filtration of 5.5 mm. Al, with the photoelectric intensitometer adjusted to have a spectral response similar to that of films exposed with intensifying screens and with scattered radiation reaching the intensitometer effectively reduced to zero. The symbols *A* to *F* have the same significance as in Figure 10.

(*b*) *Effect of Portal Size on Contrast.* The effect of portal size on the contrast of images produced by objects separated from the film or screen by more than a few

* For a more exhaustive treatment of this subject the reader is referred to a recent communication by Victoreen.[7]

TABLES XIV–XIX

MASS ABSORPTION COEFFICIENTS EXPRESSED IN TERMS OF DECADIC LOGARITHMS OF $Ca_{10}CO_3(PO_4)_6$, THE PRINCIPAL INORGANIC CONSTITUENT OF BONE. DATA ARE APPLICABLE TO CONDITIONS WHERE EXPOSURES ARE MADE WITH INTENSIFYING SCREENS

TABLE XIV

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al)

Thickness of $Ca_{10}CO_3(PO_4)_6$* (cm.)	Kilovolts (peak)			
	85	70	50	30
0.0	0.93	1.08	1.26	1.80
1.0	0.40	0.48	0.63	1.09
2.0	0.32	0.39	0.52	0.99
3.0	0.31	0.36	0.49	0.95
4.0	0.29	0.35	0.48	

* Density of material = 1.00.

TABLE XV

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al) plus 2.5 cm. Masonite presdwood

Thickness of $Ca_{10}CO_3(PO_4)_6$* (cm.)	Kilovolts (peak)			
	85	70	50	30
0.0	0.73	0.83	0.93	1.44
1.0	0.36	0.42	0.52	1.04
2.0	0.31	0.37	0.48	0.95
3.0	0.29	0.36	0.48	0.93
4.0	0.28	0.35	0.47	

* Density of material = 1.00.

TABLE XVI

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al) plus 5.0 cm. Masonite presdwood

Thickness of $Ca_{10}CO_3(PO_4)_6$* (cm.)	Kilovolts (peak)			
	85	70	50	30
0.0	0.50	0.64	0.80	1.14
1.0	0.35	0.46	0.61	0.99
2.0	0.31	0.36	0.48	0.95
3.0	0.29	0.35	0.47	
4.0	0.28	0.35	0.47	

* Density of material = 1.00.

TABLE XVII

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al) plus 10 cm. Masonite presdwood

Thickness of $Ca_{10}CO_3(PO_4)_6$* (cm.)	Kilovolts (peak)		
	85	70	50
0.0	0.41	0.50	0.69
1.0	0.32	0.38	0.52
2.0	0.29	0.36	0.48
3.0	0.28	0.35	0.47
4.0	0.28	0.35	0.47

* Density of material = 1.00.

TABLE XVIII

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al) plus 20 cm. Masonite presdwood

Thickness of $Ca_{10}CO_3(PO_4)_6$* (cm.)	Kilovolts (peak)	
	85	70
0.0	0.32	0.39
1.0	0.29	0.35
2.0	0.28	0.35
3.0	0.28	0.35
4.0	0.27	0.34

* Density of material = 1.00.

TABLE XIX

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al) plus 30 cm. Masonite presdwood

Thickness of $Ca_{10}CO_3(PO_4)_6$* (cm.)	Kilovolts (peak)	
	85	70
0.0	0.31	0.36
1.0	0.29	0.35
2.0	0.28	0.35
3.0	0.27	0.34
4.0	0.27	0.34

* Density of material = 1.00.

centimeters may be easily evaluated from Tables VI to XIII. For example, if one is roentgenoscoping a 20 cm. subject with a type B screen at 70 kv. (peak), the intensity distribution function when a grid is not used and when the portal area is 300 sq. cm. is

$$F_1 = (b_b\rho_b - b_i\rho_i) \times 0.270x_i.$$

TABLES XX–XXV

MASS ABSORPTION COEFFICIENTS EXPRESSED IN TERMS OF DECADIC LOGARITHMS OF $Ca_{10}CO_3(PO_4)_6$, THE PRINCIPAL INORGANIC CONSTITUENT OF BONE. DATA ARE APPLICABLE TO CONDITIONS WHERE EXPOSURES ARE MADE WITH INTENSIFYING SCREENS

TABLE XX

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al) plus 5.0 mm. Al

Thickness of $Ca_{10}CO_3(PO_4)_6$* (cm.)	Kilovolts (peak)		
	85	70	50
0.0	0.39	0.46	0.57
1.0	0.32	0.39	0.50
2.0	0.28	0.34	0.46
3.0	0.27	0.33	0.46
4.0	0.26	0.33	0.46

* Density of material = 1.00.

TABLE XXI

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al) plus 5.0 mm. Al plus 2.5 cm. Masonite presdwood

Thickness of $Ca_{10}CO_3(PO_4)_6$* (cm.)	Kilovolts (peak)		
	85	70	50
0.0	0.36	0.42	0.53
1.0	0.29	0.35	0.47
2.0	0.28	0.34	0.46
3.0	0.27	0.33	0.46
4.0	0.26	0.33	0.46

* Density of material = 1.00.

TABLE XXII

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al) plus 5.0 mm. Al plus 5.0cm. Masonite presdwood

Thickness of $Ca_{10}CO_3(PO_4)_6$* (cm.)	Kilovolts (peak)		
	85	70	50
0.0	0.35	0.39	0.50
1.0	0.28	0.34	0.47
2.0	0.27	0.33	0.46
3.0	0.26	0.33	0.46
4.0	0.26	0.32	0.46

* Density of material = 1.00.

TABLE XXIII

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al) plus 5.0 mm. Al plus 10 cm. Masonite presdwood

Thickness of $Ca_{10}CO_3(PO_4)_6$* (cm.)	Kilovolts (peak)		
	85	70	50
0.0	0.30	0.37	0.48
1.0	0.27	0.34	0.46
2.0	0.26	0.33	0.46
3.0	0.26	0.32	0.46
4.0	0.26	0.32	0.46

* Density of material = 1.00.

TABLE XXIV

Roentgen tube filtration = Inherent tube filtration (0.5 mm. Al) plus 5.0 mm. Al plus 20 cm. Masonite presdwood

Thickness of $Ca_{10}CO_3(PO_4)_6$* (cm.)	Kilovolts (peak)	
	85	70
0.0	0.28	0.35
1.0	0.26	0.34
2.0	0.26	0.33
3.0	0.26	0.32
4.0	0.26	0.32

* Density of material = 1.00.

TABLE XXV

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al) plus 5.0 mm. Al plus 30 cm. Masonite presdwood

Thickness of $Ca_{10}CO_3(PO_4)_6$* (cm.)	Kilovolts (peak)	
	85	70
0.0	0.27	0.34
1.0	0.26	0.33
2.0	0.26	0.32
3.0	0.26	0.32
4.0	0.26	0.32

* Density of material = 1.00.

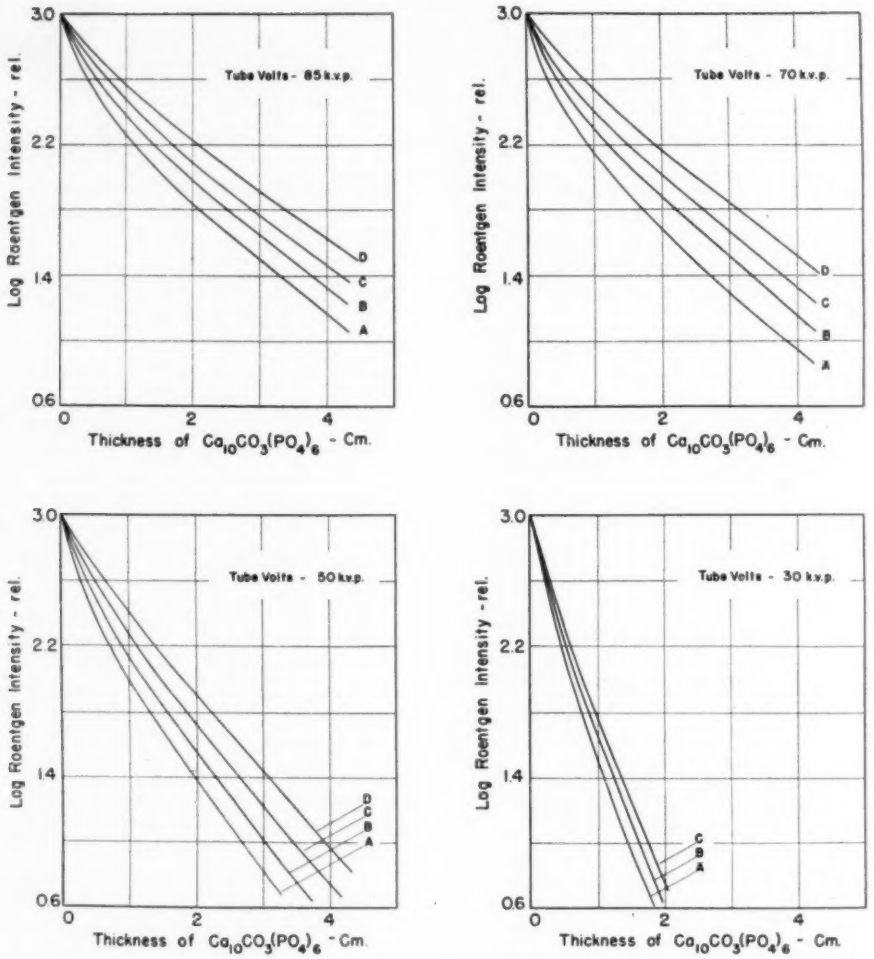

FIG. 12. Absorption curves of carbonate apatite irradiated with a four-valve, full-wave rectified generator operating at potentials of 30, 50, 70 and 85 kv. (peak), with a roentgen-ray beam filtration of 0.5 mm. Al (inherent tube filtration), with the photoelectric intensitometer adjusted to have a spectral response similar to that of films exposed with roentgen rays directly and with scattered radiation reaching the intensitometer effectively reduced to zero. Curves *A* to *D* refer respectively to conditions where additional filtration of 0.0 cm., 2.5 cm., 5.0 cm., and 10 cm., of Masonite presdwood (density = 1.07) is present.

When the portal area is 900 sq. cm. the function is

$$F_2 = (b_b\rho_b - b_i\rho_i) \times 0.135 x_i.$$

Under this circumstance, therefore, contrast is increased 100 per cent by reducing the size of the field irradiated.

(*c*) *Effect of a Grid on Contrast.* In this example let us compare the contrast, obtained in a photofluorographic film when a grid having an efficiency of 80 per cent is used, with the contrast which occurs when the grid is absent.

In the derivation let us assume a subject, 20 cm. in thickness, a roentgen tube potential of 85 kv. (peak), a type D fluorescent screen and a filtration of 0.5 mm. Al. Chamberlain[1] has pointed out that the chest of a 20 cm. subject has approximately the same absorption as 10 cm. of presdwood. Therefore, from Table XI the intensity distribution function when a grid is employed is

$$F_1 = \frac{(b_b\rho_b - b_i\rho_i) \times 0.275 x_i}{1 + 0.8(0.275 - 1.000)}$$
$$= (b_b\rho_b - b_i\rho_i) \times 0.655 x_i$$

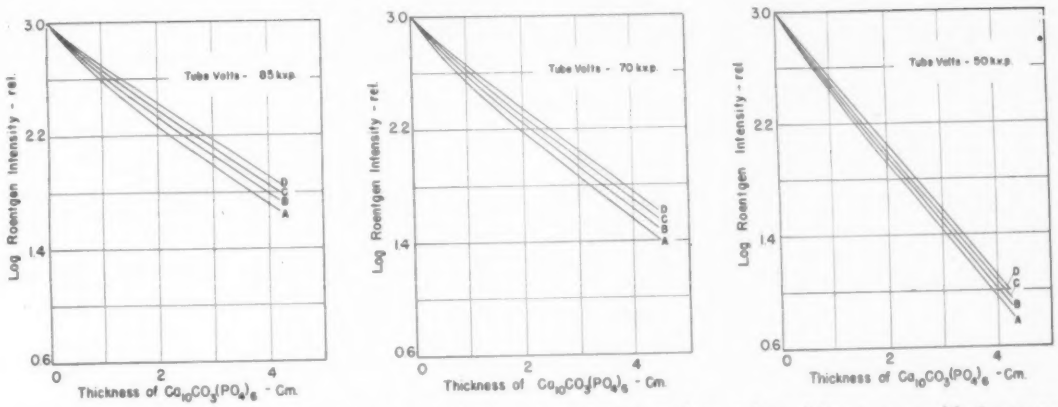

FIG. 13. Absorption curves of carbonate apatite irradiated with a four-valve, full-wave rectified roentgen generator operating at potentials of 50, 70 and 85 kv. (peak), with a total roentgen-ray beam filtration of 5.5 mm. Al, with the photoelectric intensitometer adjusted to have a spectral response similar to that of films exposed with roentgen rays directly, and with scattered radiation reaching the intensitometer effectively reduced to zero. The symbols *A* to *D* have the same significance as in Figure 12.

TABLES XXVI–XXIX

MASS ABSORPTION COEFFICIENTS EXPRESSED IN TERMS OF DECADIC LOGARITHMS OF $Ca_{10}CO_3(PO_4)_6$, THE PRINCIPAL INORGANIC CONSTITUENT OF BONE. DATA ARE APPLICABLE TO CONDITIONS WHERE EXPOSURES ARE MADE WITHOUT INTENSIFYING SCREENS

TABLE XXVI

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al)

Thickness of $Ca_{10}CO_3(PO_4)_6$* (cm.)	Kilovolts (peak)			
	85	70	50	30
0.0	1.20	1.40	1.60	1.94
1.0	0.50	0.60	0.73	1.09
2.0	0.34	0.40	0.55	0.99
3.0	0.32	0.36	0.50	0.95
4.0	0.31	0.35	0.49	

* Density of material = 1.00.

TABLE XXVII

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al) plus 2.5 cm. Masonite presdwood

Thickness of $Ca_{10}CO_3(PO_4)_6$* (cm.)	Kilovolts (peak)			
	85	70	50	30
0.0	0.88	0.99	1.15	1.67
1.0	0.44	0.50	0.59	1.05
2.0	0.34	0.38	0.53	0.95
3.0	0.32	0.36	0.50	0.93
4.0	0.31	0.35	0.49	

* Density of material = 1.00.

TABLE XXVIII

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al) plus 5.0 cm. Masonite presdwood

Thickness of $Ca_{10}CO_3(PO_4)_6$* (cm.)	Kilovolts (peak)			
	85	70	50	30
0.0	0.62	0.74	0.90	1.22
1.0	0.42	0.50	0.62	0.99
2.0	0.33	0.38	0.51	0.95
3.0	0.31	0.36	0.49	
4.0	0.30	0.35	0.48	

* Density of material = 1.00.

TABLE XXIX

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al) plus 10 cm. Masonite presdwood

Thickness of $Ca_{10}CO_3(PO_4)_6$* (cm.)	Kilovolts (peak)		
	85	70	50
0.0	0.47	0.55	0.75
1.0	0.34	0.39	0.55
2.0	0.31	0.36	0.49
3.0	0.30	0.35	0.48
4.0	0.29	0.35	0.48

* Density of material = 1.00.

Tables XXX–XXXIII

MASS ABSORPTION COEFFICIENTS EXPRESSED IN TERMS OF DECADIC LOGARITHMS OF $Ca_{10}CO_3(PO_4)_6$, THE PRINCIPAL INORGANIC CONSTITUENT OF BONE. DATA ARE APPLICABLE TO CONDITIONS WHERE EXPOSURES ARE MADE WITHOUT INTENSIFYING SCREENS

Table XXX

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al) plus 5.0 mm. Al

Thickness of $Ca_{10}CO_3(PO_4)_6$* (cm.)	Kilovolts (peak)		
	85	70	50
0.0	0.49	0.57	0.71
1.0	0.33	0.40	0.50
2.0	0.28	0.34	0.47
3.0	0.27	0.33	0.46
4.0	0.26	0.33	0.46

* Density of material = 1.00.

Table XXXI

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al) plus 5.0 mm. Al plus 2.5 cm. Masonite presdwood

Thickness of $Ca_{10}CO_3(PO_4)_6$* (cm.)	Kilovolts (peak)		
	85	70	50
0.0	0.44	0.52	0.66
1.0	0.33	0.38	0.48
2.0	0.28	0.34	0.46
3.0	0.27	0.33	0.46
4.0	0.26	0.33	0.46

* Density of material = 1.00.

Table XXXII

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al) plus 5.0 mm. Al plus 5.0 cm. Masonite presdwood

Thickness of $Ca_{10}CO_3(PO_4)_6$* (cm.)	Kilovolts (peak)		
	85	70	50
0.0	0.40	0.44	0.57
1.0	0.30	0.34	0.48
2.0	0.27	0.33	0.46
3.0	0.26	0.33	0.46
4.0	0.26	0.33	0.46

* Density of material = 1.00.

Table XXXIII

Roentgen beam filtration = Inherent tube filtration (0.5 mm. Al) plus 5.0 mm. Al plus 10 cm. Masonite presdwood

Thickness of $Ca_{10}CO_3(PO_4)_6$* (cm.)	Kilovolts (peak)		
	85	70	50
0.0	0.33	0.39	0.51
1.0	0.28	0.34	0.46
2.0	0.27	0.33	0.46
3.0	0.26	0.32	0.46
4.0	0.26	0.32	0.46

* Density of material = 1.00.

and when a grid is absent

$$F_2 = (b_b\rho_b - b_i\rho_i) \times 0.275 x_i.$$

Thus the use of a grid increases photofluorographic contrast by almost 130 per cent.

(*d*) *Effect of Filtration on Contrast.* The effect of filtration on the mass absorption coefficients of presdwood and $Ca_{10}CO_3(PO_4)_6$ depends in a large measure on the thickness of the phantom under examination, being quite marked when the thickness is small and rather insignificant when the thickness is large. The manner in which contrast is governed by filtration will therefore be very different under the two conditions.

Let us first consider the image contrast produced by a calcified object having a density of 2.0 and a thickness of 0.25 cm. when surrounded by skeletal muscle having a density of 1.05 and a thickness of 10 cm. If the field irradiated is 300 sq. cm., the roentgen beam filtration is 0.5 mm. Al, the roentgen tube potential is 70 kv. (peak) and a grid having an efficiency of 80 per cent is used, the intensity distribution function of the image when recorded by a film exposed with intensifying screens is

$$F_1 = \frac{(0.113 \times 1.02 \times 1.05 - 0.50 \times 2.0)0.25 \times 0.475}{(1 + 0.8(0.475 - 1.000)} = 0.24.$$

When the filtration is 5.5 mm. Al the function is

$$F_2=\frac{(0.107\times 1.02\times 1.05-0.37\times 2.0)0.25\times 0.350}{1+0.8(0.350-1.000)}$$
$$=0.11.$$

Thus the introduction of filtration reduces the contrast of the image by almost 55 per cent.

If, however, the calcified object is surrounded by muscle tissue having a thickness of 20 cm. and the size of the field irradiated is 900 sq. cm., the intensity distribution function of the object's image when the roentgen beam filtration is 0.5 mm. Al will be

$$F_1=\frac{(0.109\times 1.02\times 1.05-0.39\times 2.0)0.25\times 0.135}{(1+0.8(0.135-1.000)}$$
$$=0.072$$

and when the filtration is 5.5 mm. Al,

$$F_2=\frac{(0.107\times 1.02\times 1.05-0.35\times 2.0)0.25\times 0.120}{(1+0.8(0.120-1.000)}$$
$$=0.058.$$

Under this second circumstance the contrast of the image is reduced by the relatively insignificant amount of 19 per cent by the added filtration.

In all of the foregoing examples it has been assumed that the scattered radiation produced by Masonite presdwood is identical to that developed by comparable thicknesses of human tissue. In view of the similarity in the absorption characteristics of presdwood, fat and skeletal muscle this assumption seems justified, although its validity will require checking by further experimentation.

It is hoped that the information which has been presented in this communication will constitute a useful guide to those who wish quantitative knowledge of the manner in which the contrast of a roentgen image is influenced by various technical factors. The data which have been given are far from complete, but additions may be expected when further investigations are completed. Meanwhile many roentgen technical problems may find ready solution from the values listed in the several tables and graphs.

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EDITORIALS

THE SIGNIFICANCE OF THE VOLUME DOSE

DURING the course of irradiation of a given lesion by means of roentgen or radium rays a certain amount of the dose administered is absorbed throughout the entire body. This fact was recognized from the earliest days of radiation therapy and references to it repeatedly appeared in the literature. However, due to lack of measuring devices, the magnitude of the dose absorbed beyond the limits of the treated area could not be exactly determined. It is not surprising, therefore, that the clinical interpretation of the effect of such a dose on the various physicochemical reactions of the body, especially in relation to radiation sickness, should have lead to considerable argumentation and speculation.

In 1935 it occurred to Ellis[6] that some advantage may be derived from trying to express the dose absorbed beyond the treated area in some sort of a unit that could be measured at least approximately. He coined the term "volume dose" although the same term was used, in a more general sense, by Wintz earlier, and described it as so many roentgens per cubic centimeter of body tissue or "roentgen cc."

In 1940 Mayneord,[1] after a series of experimental and theoretical investigations, placed the subject on a more solid physical foundation. He suggested the term "integral dose" and introduced as a basic unit the gram-roentgen which is the energy conversion when 1 r is delivered to 1 gram of air. A megagram-roentgen (1 million gram-roentgens) is approximately equivalent to 2 calories. In radium therapy results are expressed in gram-roentgens per milligram-hour. Although this unit of Mayneord, in assuming unity density of the patient, refers to weight rather than volume, it has distinct clinical advantages. It makes, above all, possible the expression of the total dose absorbed by the body during the course of irradiation in a manner comparable with the absorption of a gram of drug per body weight.

Following the initial work of Ellis and of Mayneord, other notable publications made their appearance in rapid succession. They dealt with the various phases of the problem by using several comparative methods of investigation. Clinical interpretations and practical evaluation of the physical data made available were likewise not slow in forthcoming.

It is, therefore, very fortunate indeed that the British Institute of Radiology, at one of its general meetings has arranged a symposium[2] aiming to co-ordinate the latest views on the physical, biochemical, and therapeutic aspects of the volume dose.

In this symposium, Clarkson[3] discussed the results of the physical measurements made in conjunction with Mayneord on a "wax" patient. A model of elliptical cross-section and divided into slabs was constructed. In some of these slabs condenser ionization chambers were housed and measurements made for various locations. The investigations included whole body irradiation, in which the tube was placed at a distance of 250 cm. to cover the entire model, and limited field irradiations. By taking the average doses and making use of a simple calculation devised by Mayneord, tables were drawn up for whole body and different kinds of trunk irradiations. The volume dose is then found by reading the average dose from these tables and multiplying it by the weight of the patient and the surface dose.

Boag,[4] on the other hand, presented the results of the physical measurements made in conjunction with Walter on a "celluloid" patient devised by Grimmett. This model

was constructed of cellulose acetate plates 6 mm. thick, spaced apart by washers of the same material 2 mm. thick. The plates were graphited to make them electrically conductive. Alternate plates were connected together with wires which were led to opposite poles of a battery. A sensitive galvanometer in the circuit measured the total ionization current collected from the air gaps when these were in a radiation field. As may be seen, such a model lends itself particularly to the measurement of the volume dose emanating from the irradiation of limited fields of the entire body. In this respect it is complementary to the "wax" patient which permits only whole body or trunk measurements.

Boag studied a number of situations with roentgen rays of 2 to 4 mm. Cu half-value layer, and found that the volume dose depends principally on the size of area and the site of the field and to a much lesser extent on the focal skin distance, linear dimensions of the patient and, within the ranges mentioned, on the half-value layer of the radiation.

The biochemical aspect of the volume dose is as yet unexplored, although, according to Holmes,[5] the data available in a few cases allow rather clear suggestions for future work. The liberation of histamine and the behavior of purines following irradiation may prove of some significance. It was noted with considerable interest that uric acid, which is the end product of purine metabolism, appeared in animal tumor cells in increasing amounts twenty-four hours after irradiation.

A great deal more is known, however, about the clinical aspect of the volume dose. Ellis[6] by reviewing the literature and collecting all the pertinent material on the subject was able to formulate rather definite conclusions as to its value in practical radiation therapy.

Ellis paid special attention to the relationship of the volume dose and a given tumor dose. The aim in clinical therapy is to administer the maximum amount of radiation to a tumor by producing the minimum possible effect on the surrounding structures or the body as a whole. Therefore, any procedure that can bring about this desideratum is worthy of consideration. Ellis found that a knowledge of the physical factors permits the advantageous adjustment of the relationship between volume dose and tumor dose in several ways. First, there is an almost proportional dependence of the volume dose on the size of the irradiated field. This means that, other things being equal, it is desirable to irradiate with fields as small as possible under the circumstances in order to keep the volume dose to a minimum value. Second, there is an inverse ratio between the volume dose and the skin target distance. This, in turn, means that to deliver the same tumor dose, other things being equal, the greater the skin target distance, the smaller the volume dose and thus the less the untoward effect from which the patient will suffer. Third, there is a definite dependence of the volume dose on the quality of the irradiating beam. Within the ranges of 2 to 4 mm. Cu half-value layer studied by Boag the difference was rather small. But in comparing 200 kv. radiation with 1,000 kv. radiation Phillips[7] found that the volume dose is greater with the higher voltage beam for the same surface dose, other things remaining equal, although it is possible by adjusting the size and number of fields, to gain an advantage with the 1,000 kv. over the 200 kv. radiation. Fourth, the volume dose can be brought to a minimum in a given case by a proper selection of the number of fields and their efficient arrangement, other things being equal. This phase of the problem was studied in extenso by Ungar.[8] He chose various set-ups for treatment of the same lesion and determined the relation of the "economy quotient" to the volume dose. The economy quotient was defined as the ratio of the minimum tumor dose divided by the difference between the maximum and minimum tumor doses. It was found that, other things being equal, the set-up which gave the greater economy quotient gave the smaller volume dose. From this Ungar devised a number of practical set-ups which he considered op-

timum for certain lesions under well determined circumstances.

However exacting and precise these physical considerations may be, the evaluation of the biologic phenomena directly attributable to the effect of the volume dose constitutes a most difficult problem. As Ellis so vividly states, human beings, unlike Mayneord's or Grimmett's creations, are complex physicochemical factories subject to mental and a multitude of pathologic influences acting in different individuals in very different ways. Therefore, any simple correlation of physical factors and clinical reactions appears practically impossible.

Broadly speaking, the biologic phenomena produced by the volume dose are general as distinct from local reactions which are the result of direct irradiation. According to Ellis, they may be divided into subjective and objective. The former include malaise, nausea, vomiting, headache, etc., all of which are impossible to measure or properly evaluate beyond the rough estimation that a larger volume dose is more apt to make a person ill than a smaller dose. To the latter belong the change in the blood pattern and some other changes, as for example those observed in the corpuscular volume, color index, sedimentation rate and blood pressure. The greatest hope was placed on the study of the blood pattern changes. Bush[9] in 1943 published a curve expressing the proportional decrease of the lymphocytes in percentage of the initial count with the increase of the volume dose. Other investigators published similar curves for all types of leukocytes. However, Ellis, in examining the blood counts in a series of patients with the same view in mind, failed to find such a close correlation between the lymphocytes and still less between the other types of leukocytes and the volume dose. The same may be said of the other measurable quantities mentioned above.

The following conclusions may therefore be derived as to the significance of the volume dose in present day therapy. The physical considerations are based on sound principles and the procedures are accurate. They aid considerably in devising methods of practical application of the roentgen and radium rays, which enable us to deliver a certain dose with greatest efficiency to the lesion, and least untoward effect to the patient. They also help in outlining methods of protection. For example, Clarkson demonstrated that a person working in a deep roentgen therapy department may absorb a volume dose which is five times as large for an equal measured stray radiation dose as that absorbed by a person engaged in roentgen diagnosis. But the volume dose is a disappointing and unreliable guide of the biologic effects. Ellis states that even if the lymphocyte curve could be correlated with the volume dose it is safer to trust the lymphocyte count rather than the physical magnitude of the volume dose in deciding when to stop treatment.

It is possible that the future will hold greater promise. In the megavoltage roentgen therapy domain where the depth doses often represent multiple values of the surface dose, the volume dose may acquire noteworthy significance. Likewise, in the therapeutic application of artificial radioactive substances the volume dose may prove a convenient indicator of the maximum tolerance under well determined circumstances.

T. Leucutia

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WALTER BRADFORD CANNON
1871-1945

DR. WALTER BRADFORD CANNON, an honorary member of the American Roentgen Ray Society since 1913, died on October 1, 1945.

In 1932 the Harvard University Press published a small volume entitled "Walter Bradford Cannon: Exercises Celebrating Twenty-five Years as George Higginson Professor of Physiology. October 15, 1931." In addition to the lengthy list of honors, publications to date, and a reproduction of the portrait accepted for the University by President Lowell, this book records the tributes paid to Dr. Cannon by Alvarez, Edsall, Lusk, and Howell. In his response, Dr. Cannon expressed some ideas which have been expanded in "The Way of an Investigator," his autobiography which is

obtainable today even in book-stores for the laity.

It was my good fortune to have "The Mechanical Factors of Digestion," then only two years published, assigned to me in 1913 to summarize for the student seminars which Dr. Sutherland Simpson exacted of his freshman medical students in physiology. It was my belief that because of the lead protection about the tube employed very early in his researches, Cannon had been spared the injuries of the other roentgen-ray pioneers. In the words of Cannon's classmate, Percy Brown, "nothing could be farther from the truth." In *Science* for November 9, 1945, Dr. Cecil K. Drinker states: "Fifteen years ago, Dr. Cannon learned he was afflicted by one of the inexorable lymphomatous diseases, all so poorly understood and all so disastrous. His illness inevitably ate a little into his humor and his irrepressible good spirits, but not into his will to work, and only those nearest to him could realize a vestige of change." It is certainly not apparent to the uninitiated reader of the Harvard 1932 volume that even as Cannon's colleague Lusk was wishing him continued health to carry on his work, Cannon knew that he was a victim of lymphoma.

If in this obituary, coming from one who from his first year in medicine on has deeply appreciated Cannon's position as Titan among physiologists, little is said of Cannon the man and much is said of his last illness, it is for a reason. Cannon's biography is on record in the references cited, and his position is such, as the founder of the art of gastrointestinal roentgen examination, that every radiologist owes it to himself and to his work, to own and to read Cannon's autobiography. A further reference is offered, to Cannon's Caldwell Lecture published in this JOURNAL for November, 1934, prefaced by the introductory remarks that only Percy Brown can make.

But there is very little in Cannon's own writings that bears on his roentgen-ray injuries. Probably no one identified with medicine ever made more extensive personal contributions to the advancement of knowledge, to say nothing of the thirty-one professors of physiology and allied subjects whom he had trained up to 1931. When such a life ends under circumstances that in themselves advance the knowledge of a subject still in the stage of collecting evidence, Percy Brown and I have come independently to the conclusion that the story should be told. This is the sequence of events: In the autumn of 1896, Professor Henry P. Bowditch suggested to Cannon that he might utilize the newly discovered roentgen ray to study the process of digestion in animals. The very beginning of gastrointestinal roentgen diagnosis was when Cannon fed a bolus of mush mixed with bismuth to a goose, and watched its progress down the esophagus. (Transit time, twelve seconds.) Late in December Cannon demonstrated this phenomenon to the American Physiological Society, and Graham Lusk recalled it thirty-five years later, remarking that he told the story of the demonstration to every class he taught in physiology.

In the Caldwell Lecture aforementioned, Cannon says: "Well do I recall the sage advice of Professor Henry P. Bowditch, when, in the spring of 1897, I showed him an area on my hand from which the skin, exposed repeatedly to the strange new rays, had come away in successive layers. He remarked that we were employing a quite novel agent which might have very dangerous properties and that we ought to be cautious until we learned more about its nature. At the time I was an eager first-year medical student, not pleased with being checked in my purposes. In order to avoid further harm I surrounded the tube with a metal box, leaving a small aperture at the top through which the rays could pass. . . . Consequently today [1934] only a few scars remain, although during a decade, I often sat for hours together watching with the fluoroscope the processes of digestion in different parts of the alimentary canal."

After the tube was enclosed with lead except for a small aperture, Cannon worked so long with fluoroscopy that even scattered radiation must needs add up. Dr. Merrill C.

Sosman has written me as follows: "Dr. Cannon had rather severe, very painful x-ray burns on the ulnar side of each hand and wrist, extending onto the fifth finger, obviously acquired while holding the fluoroscopic screen with his hands on edge. These required several excisions and skin grafts and they bothered him for years, up to the time of his death. Not many men knew this and he never complained publicly; but I thought you should know it. For about fifteen years he suffered intense itching from leukemia cutis (mycosis fungoides), relieved only by roentgen treatments, and his death was eventually due to malignant lymphoma, not leukemia as reported in the *Journal of the American Medical Association*. He was one of the most beloved of the Harvard faculty, a friend and adviser to all of us, and we shall miss him sadly."

Percy Brown writes: "Walter suffered much injury as a result of his early work. I remember well the occurrences of acute dermatitis along the inside of each thigh (in much of his work his tube-container was suspended practically between his legs). This was many years ago, but more recently, in fact but a very few years before his death, he underwent an operation for the excision of an epitheliomatous lesion of his lip, from which he had gotten, apparently, good surgical results.

"Walter and I were class-mates, and I have followed the course of his life with the interest born of devoted friendship. I saw him last during the month of July, when we were both in the Massachusetts General Hospital, I for an amputation, and he for an acute exacerbation of leukemia cutis, with its intolerable itching, and its purpuric manifestations. His suffering for some time had been intense, especially since the weather was frightfully hot. I was conveyed in my wheel-chair to his room, and I marvelled at the degree of his fortitude and patience. He remained in the hospital a short time after my discharge, and was then transferred to his summer home in New Hampshire, whence I received letters from him. He knew, of course, the inevitable prognosis of his case, and faced the outcome with his characteristic courage."

Cannon entered Harvard University with one hundred and eighty dollars. He earned his way through the University and the Medical School, and emerged with an earned balance of seven hundred dollars. On June 25, 1901, he was married to Miss Cornelia James, who as an author is mentioned in her own right in *Who's Who in America*. This most happy marriage was blessed with a son, Bradford, and four daughters, Wilma Denio, Linda, Maria and Helen. Cannon found great joy in his children. When urged to work a little less hard, he took up sculpture, and modeled a head of his daughter, which took a blue ribbon in an exhibition of physician-artists. Drinker observes, "He was better than a good sculptor." Of all the honors which Cannon received, he remarked that none ever gave him more satisfaction than the "eight inches of blue ribbon" awarded to this creation. Certainly he could not have been less gratified by A. J. Carlson's review of "The Way of an Investigator," which, appearing in *The Scientific Monthly* for October, 1945, he did not live to see in print. The italics are Carlson's: "These chapters should be *must-reading for all medical students* if not all students in science in college and high school. This volume is not above the comprehension of laymen, and wide reading of Dr. Cannon's story would broaden the base of real understanding of science in our beloved land."

To resume the letter from Percy Brown: "Since his death, Cornelia has written me: 'Dear Percy, Your sweet letter comforted me. It is a strength to feel the friends gather close. Walter, like you, proved to be a martyr to the work with the x-rays. Two weeks before he died, he gave a very fine address before the Shock Committee. Then he had a normal week when we both felt he was on the road to health, when his dread disease overtook him, and after a week more or less, of coma, he died peacefully'."

RAMSAY SPILLMAN

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. H. Dabney Kerr, University Hospital, Iowa City, Iowa. Annual meeting: Netherland Plaza Hotel, Cincinnati, Ohio, Sept. 17–20, 1946.

American College of Radiology
Secretary, Mac F. Cahal, 540 N. Michigan Ave., Chicago.

Section on Radiology, American Medical Association
Secretary, Dr. U. V. Portmann, Cleveland Clinic, Cleveland, Ohio. Annual meeting: San Francisco, Calif., July 1–5, 1946.

Arkansas Radiological Society
Secretary, Dr. J. S. Wilson, Mack Wilson Hospital, Monticello, Ark. Meets every three months and also at time and place of State Medical Association.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: 1946, to be announced.

Radiological Section, Baltimore Medical Society
Secretary, Dr. Walter L. Kilby, Baltimore. Meets third Tuesday each month, September to May.

Section on Radiology, California Medical Association
Secretary, Dr. Gordon G. King, 3700 California St., San Francisco 18, Calif.

Radiological Section, Connecticut Medical Society
Secretary, Dr. Max Climan, 242 Trumbull St., Hartford, Conn. Meets bi-monthly on second Thursday, at place selected by Secretary. Annual meeting in May.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. H. W. Ackemann, 321 W. State St., Rockford, Ill.

Radiological Section, Los Angeles County Medical Association
Secretary, Dr. Roy W. Johnson, 1407 S. Hope St., Los Angeles, Calif. Meets on second Wednesday of each month at the County Society Building.

Radiological Section, Southern Medical Association
Secretary, Dr. Roy G. Giles, Temple, Texas.

Brooklyn Roentgen Ray Society
Secretary, Dr. Leo Harrington, 880 Ocean Ave., Brooklyn, N.Y. Meets monthly on fourth Tuesday, October to April.

Buffalo Radiological Society
Secretary, Dr. Joseph S. Gian-Francheschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months.

Chicago Roentgen Society
Secretary, Dr. F. H. Squire, 1754 W. Congress St., Chicago 12, Ill. Meets second Thursday of each month October to April inclusive at the Palmer House.

Cincinnati Radiological Society
Secretary, Dr. Samuel Brown, 707 Race St., Cincinnati, Ohio. Meets third Tuesday of each month, October to May, inclusive.

Cleveland Radiological Society
Secretary, Dr. Carroll C. Dundon, 2065 Adelbert Road, Cleveland 6, Ohio. Meetings at 6:30 P.M. on fourth Monday of each month from October to April.

Dallas-Fort Worth Roentgen Study Club
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meetings held in Dallas on odd months and in Fort Worth on even months, on third Monday, at 7:30 P.M.

Denver Radiological Club
Secretary, Dr. A. Page Jackson, Jr., 1612 Tremont Place, Denver, Colo. Meets third Friday of each month at Denver Athletic Club.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

Florida Radiological Society
Secretary, Dr. J. F. Pitman, Blanch Hotel Annex, Lake City, Fla. Meetings in May and November.

Georgia Radiological Society
Secretary, Dr. James J. Clark, 478 Peachtree St., Atlanta, Ga. Meets in November and at annual meeting of Medical Association of Georgia in the spring.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Illinois Radiological Society
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

Indiana Roentgen Society
Secretary, Dr. H. C. Ochsner, Methodist Hospital, Indianapolis. Meets annually second Sunday in May.

Iowa X-Ray Club
Secretary, Dr. Arthur W. Erskine, 326 Higley Bldg., Cedar Rapids, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. Special meetings by announcement.

Kentucky Radiological Society
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in Apr.

Long Island Radiological Society
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:30 P.M.

Louisiana Radiological Society
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

Michigan Association of Roentgenologists
Secretary, Dr. E. M. Shebesta, 1429 David Whitney Bldg., Detroit. Three meetings a year, Fall, Winter, Spring.

Milwaukee Roentgen Ray Society
Secretary, Dr. C. A. H. Fortier, 231 W. Wisconsin Ave., Milwaukee, Wis. Meets monthly on second Monday at University Club.

Minnesota Radiological Society
Secretary, Dr. Annette T. Stenstrom, 1218 Medical Arts Bldg., Minneapolis, Minn. One meeting a year at time of Minnesota State Medical Association.

Nebraska Radiological Society
Secretary, Dr. D. A. Dowell, Medical Arts Bldg., Omaha, Nebr. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

New England Roentgen Ray Society
Secretary, Dr. George Levene, Massachusetts Memorial Hospitals, Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

New Hampshire Roentgen Ray Society
Secretary, Dr. Richard C. Batt, Berlin, N. H. Four meetings a year.

Radiological Society of New Jersey
Secretary, Dr. H. R. Brindle, 501 Grand Ave., Asbury Pk. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

New York Roentgen Society
Secretary, Dr. Ramsay Spillman, 115 East 61st St., New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:30 P.M.

North Carolina Roentgen Ray Society
Secretary, Dr. Major Fleming, Rocky Mount, N. C. An-

* Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

nual meeting at time and place of State Medical Society. Mid-year scientific meeting at place designated.

North Dakota Radiological Society
Secretary, Dr. L. A. Nash, St. John's Hospital, Fargo. Meetings held by announcement.

Central New York Roentgen Ray Society
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year. January, May, November.

Ohio Radiological Society
Secretary, Dr. Henry Snow, 1061 Reibold Bldg., Dayton, Ohio. Meets during annual meeting of Ohio State Medical Association.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. L. E. Wurster, 416 Pine St., Williamsport.

Philadelphia Roentgen Ray Society
Secretary, Dr. C. L. Stewart, Jefferson Hospital, Meetings first Thursday of each month, October to May, at at 8:00 P.M., in Thomson Hall, College of Physicians, 21 S. 22d St.

Pittsburgh Roentgen Society
Secretary, Dr. L. M. J. Freedman, 4800 Friendship Ave. Meets 6:30 P.M. at The Ruskin on second Wednesday, each month, October to May inclusive.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Murray P. George, Strong Memorial Hospital. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary Dr. A.M. Popma, 220 N. First St., Boise, Idaho.

St. Louis Society of Radiologists
Secretary, Dr. Edwin C. Ernst, Beaumont Medical Building, St. Louis, Mo. Meets fourth Wednesday of each month, except June, July, August, and September, at a place designated by the president.

San Diego Roentgen Society
Secretary, Dr. Henry L. Jaffe, Naval Hospital, Balboa Park, San Diego, Calif. Meets monthly on first Wednesday at dinner.

San Francisco Radiological Society
Secretary, Dr. Carlton L. Ould, University of California Hospital, San Francisco 22. Meets monthly on the third Thursday at 7:45 P.M., first six months of the year at Lane Hall, Stanford University Hospital, and second six months at Toland Hall, University of California Hospital.

Shreveport Radiological Club
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

South Carolina X-Ray Society
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

Tennessee Radiological Society
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth 4, Texas. Next meeting, Dallas, Texas, Monday, January 14, 1946.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets every Thursday from 4:00–5:00 P.M., Room 301, Service Memorial Institute.

Virginia Radiological Society
Secretary, Dr. E. L. Flanagan, 116 E. Franklin St., Richmond, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Thomas Carlile, 1115 Terry St., Seattle. Meets fourth Monday each month, October through May, College Club, Seattle.

X-ray Study Club of San Francisco
Secretary, Dr. J. M. Robinson, University of California Hospital. Meets monthly, third Thursday evening.

Cuba

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Medical Members' meeting held monthly on third Friday at 2:30 P.M. and Ordinary Meeting at same time on following Saturday, October to May, 32 Welbeck St., London, W.1.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets on the third Friday of each month at 4:45 P.M. at the Royal Society of Medicine 1, Wimpole St., London, W. 1.

Faculty of Radiologists
Secretary, Dr. M. H. Jupe, 32 Welbeck St., London, W. 1 England.

Section of Radiology and Medical Electricity, Australasian Medical Congress
Secretary, Dr H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

Radiological Section of the Victorian Branch of the British Medical Association
Secretary, Dr. Keith Hallam, St. George's Hospital, K.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to November inclusive.

Canadian Association of Radiologists
Secretary, Dr. J. W. McKay, 1620 Cedar Ave., Montreal, P. Q.

Société Canadienne-Francaise d'Electrologie et de Radiologie Médicales
Secretary, Dr. Origène Dufresne, 4120 Ontario St., East Montreal, P. Q.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Radiological Section, New Zealand British Medical Association
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

South America

Sociedad Argentina de Radiologia
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

Sociedad Peruana de Radiologia
Secretary, Dr. Victor Giannoni, Apartado, 2306, Lima, Peru. Meetings held monthly except during January, February and March, at the Asociación Médica Peruana "Daniel A. Carrión, Villalta 218, Lima.

Continental Europe

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Société Suisse de Radiologie (Schweizerische Röntgen-Gesellschaft)
Secretary for French language, Dr. Babaiantz, Geneva. *Secretary* for German language, Dr. Max Hopf, Effingerstrasse 49, Bern. Meets annually in different cities.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banul Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad: USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 P.M.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

WATTS HOSPITAL MEDICAL AND SURGICAL SYMPOSIUM

The Third Annual Watts Hospital Medical and Surgical Symposium will be held at the Watts Hospital, Durham, North Carolina, on Wednesday and Thursday, February 13 and 14, 1946. The following is the program to be presented:

Wednesday, February 13, 1946

10:30 A.M. W. R. Stanford, M.D., presiding. Address of Welcome. Mr. Sample B. Forbus, Superintendent, Watts Hospital.

10:35 A.M. Greetings from the Board of Trustees of Watts Hospital, Mr. George Watts Hill, President.

10:45 A.M. Some Remarks on Postgraduate Medicine. W. C. Davison, M.D., Dean, Duke University Medical School, Durham, N.C.

11:00 A.M. F. N. Bowles, M.D., presiding. Clinico-Pathological Conference. William B. Porter, M.D., Professor of Medicine, Medical College of Virginia, and John S. Howe, M.D., Professor of Pathology, Medical College of Virginia, Richmond, Va.

1:00 P.M. Luncheon, Washington Duke Hotel.

2:30 P.M. W. W. Vaughan, M.D., presiding. Some Hemolytic Processes Including the Rh Factor. Lt. Col. Louis Krause, Chief of Medicine, General Hospital, Camp Butner, North Carolina.

3:30 P.M. The Treatment of Habitual Abortion. Norris W. Vaux, M.D., Professor of Obstetrics, Jefferson Medical College, Philadelphia, Pa.

6:00 P.M. Barbecue Dinner at Watts Hospital, compliments of the Hospital Administration.

8:00 P.M. William M. Coppridge, M.D., presiding. Psychiatry Speaks to Democracy—About Mothers and Moms. Edward A. Strecker, Professor of Psychiatry, University of Pennsylvania Medical School, Philadelphia, Pa.

9:00 P.M. Diverticulitis of the Colon. Arthur M. Shipley, M.D., Professor of Surgery, University of Maryland Medical School, Baltimore, Md.

Thursday, February 14, 1946

11:00 A.M. J. B. Bullitt, M.D., presiding. Clinico-Pathological Conference. Henry B. Mulholland, M.D., Professor of Medicine, University of Virginia Medical School, and James R. Cash, M.D., Professor of Pathology, University of Virginia Medical School, Charlottesville, Va.

1:30 P.M. Luncheon, Washington Duke Hotel.

2:30 P.M. A. H. Powell, M.d., presiding. The Relationship of Medical Practice to Gerontology. James E. Paullin, M.D., Professor of Medicine, Emory University Medical School, Atlanta, Ga.

3:30 P.M. General Features of Periarteritis Nodosa, particularly from the Standpoint of Diagnosis. Louis Hamman, M.D., Associate Professor of Medicine, Johns Hopkins Medical School, Baltimore, Md.

6:00 P.M. Dinner, Washington Duke Hotel.

8:00 P.M. Hunter Sweaney, M.D., presiding. The Pathologic Physiology of Biliary Tract Disease. I. S. Ravdin, Professor of Surgery, University of Pennsylvania Medical School, Philadelphia, Pa.

8:30 P.M. The Roentgenological Aspects of Biliary Tract Disease. Eugene P. Pendergrass, M.D., Professor of Radiology, University of Pennsylvania Medical School, Philadelphia.

9:30 P.M. The Surgical Aspects of Gallstone Disease. I. S. Ravdin, M.D.

AMERICAN COLLEGE OF PHYSICIANS

The American College of Physicians will resume its annual meetings in 1946 and has chosen Philadelphia as the meeting place and the dates May 13 to 17 inclusive. Headquarters will be at the Philadelphia Municipal Auditorium, 34th Street below Spruce. The meeting will be conducted under the Presidency of Dr. Ernest E. Irons, Chicago, Illinois, and the General Chair-

manship of Dr. George M. Piersol, Philadelphia, Pennsylvania.

BOSTON MEDICAL HISTORY CLUB

The Boston Medical History Club held its third meeting of the season on Monday, December 10, 1945, at 8:15 P.M., at the Boston Medical Library. The meeting commemorated the centennial of the birth of Röntgen and the semicentennial of his discovery of the roentgen ray. The speaker of the evening was Dr. Merrill C. Sosman, Clinical Professor of Radiology, Harvard Medical School. An exhibition of books was arranged by Mr. James F. Ballard, Director, Boston Medical Library.

TEXAS RADIOLOGICAL SOCIETY

The annual meeting of the Texas Radiological Society will be held on Monday, January 14, 1946, in Dallas, Texas. All of the sessions and the annual banquet on Monday evening will be held at the Baker Hotel.

DEPARTMENT OF TECHNIQUE

Department Editor: ROBERT B. TAFT, M.D., B.S., M.A., 103 Rutledge Ave. Charleston, S. C.

AN APPARATUS FOR THE UTILIZATION OF WEAK RADON TUBES

By L. CARDENAS, E.E., *and* J. L. WEATHERWAX, M.A.
Philadelphia General Hospital
PHILADELPHIA, PENNSYLVANIA

THE practice of using radon tubes in radiation therapy generally results in the discarding of those tubes which have decayed to 20 millicuries or less. This is to avoid the undue lengthening of the dosage time for clinic patients.

In order to utilize these tubes for the preparation of gold seeds, a vacuum apparatus was developed at the Philadelphia General Hospital. This apparatus has been in use for approximately a year and a half and has given satisfactory service. The section as shown in Figure 1 can be constructed as a separate unit with a suitable evacuating system or can be attached to the Failla-Duane emanation plant.[1] The radon is transferred from the crusher into the gold capillary by means of the usual Toepler mercury pumping system.

The idea of crushing weak radon tubes is not original with the authors. Dr. G. Failla at Memorial Hospital, New York, in 1934 devised a special stopcock for this purpose. Its use was discontinued in 1936 owing to a number of reasons, chief of which was the necessity of glassblowing and the exposure to which the technician was subjected in turning the stopcock by hand. These difficulties are overcome in the solenoid operated crusher used at the Philadelphia General Hospital.

The crusher tube shown on the right of Figure 1 is made of a thick-walled pyrex tube to insure mechanical strength. The bottom of this tube is rounded and the upper end is tapered and ground to fit over a hollow plug of soda glass which in turn is sealed to the bottom of a soda glass grease stopcock. This was done to avoid the use of a graded seal since emanation plants are generally constructed of soft glass tubing. Ordinary stopcock grease is used as a lubricant. It was found that the greased stopcock functions without excessive gassing due to the shortness of contact time with the radon. Inside the pyrex cylinder and resting on the bottom is a small solid brass plug. The lower end of this plug is rounded and

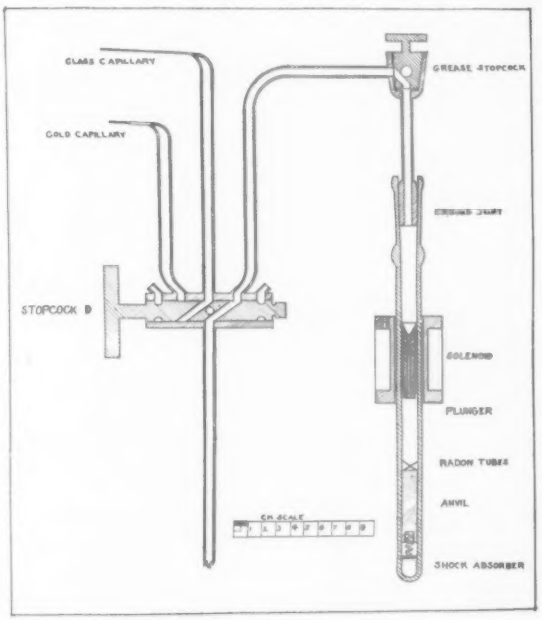

FIG. 1.

[1] Failla, G. U. S. Patents 1,553,794; 1,609,614.

the upper end is flat and recessed to hold a small vertical spring. A solid brass cylinder having a flat upper surface rests on the spring. This serves as the "anvil" of the crusher. The plunger is a hollow soft iron cylinder. Its upper end is countersunk to a 60 degree angle and the bottom turned down or reduced in cross section as shown.

Figure 2 illustrates the manner in which the solenoid crusher C_5 is appended to the Failla-Duane emanation plant. Also shown in this figure is a degassing chamber C_4 used by us in an experimental investigation.

The procedure is to admit air into stopcock D and hence also into the crusher. The plug of the greased stopcock is removed and

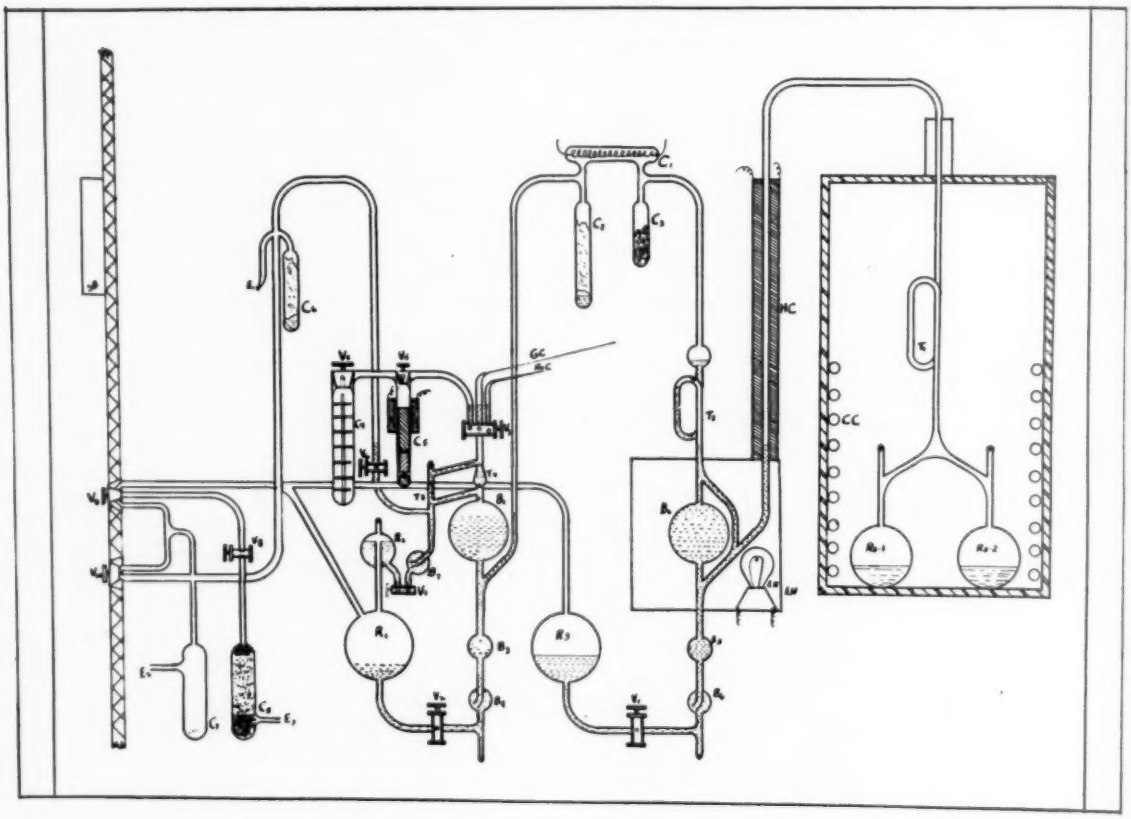

FIG. 2. Radium emanation plant, Philadelphia General Hospital; Failla-Duane type.

The lower portion of the crusher tube is enclosed in a concentric lead cylinder (not shown in the figure). This shield has a hole bored through it transversely and is illuminated from the rear. Thus the crushing of the tubes may be viewed without exposing the technician to radiation. Above the shield and also concentric with the crusher tube rests an electromagnetic coil. The entire assembly is clamped to the frame of the radon extraction apparatus to provide rigid support.

a glass funnel inserted in its place. The glass tubes which contain radon are removed from their filtration jackets and are placed in ether to remove any adherent paraffin. When they are clean and dry they are dropped through the funnel, the neck of which extends deep into the crusher. The tubes then come to rest within the hollow soft iron plunger. The funnel is removed and the plug of the greased stopcock inserted, and rotated to assure a good seal. The stopcock D and the crusher are then

evacuated. When a vacuum has been obtained the remainder of the system is isolated from stopcock *D* and the crusher. The solenoid is energized by an electrical current and the tubes fall through the hollow soft iron plunger and come to rest in an oblique position on the "anvil" as indicated in the sketch. By energizing and de-energizing the solenoid two or three times the plunger crushes the tubes to powder liberating the radon. Due to the high atomic weight of the radon and the small bore of the greased stopcock a little time should be allowed for the diffusion of the radon into the bulb of the radon apparatus directly below stopcock *D*. At the Philadelphia General Hospital it has been found that a single "stroke" will transfer 65 to 70 per cent of the radon into the gold capillary or into the glass capillary. A higher percentage of transfer is secured by taking a greater number of "strokes."

It may be found, possibly due to slight gassing of the grease or the presence of moisture or both, that the application of one-half atmosphere of pressure will be insufficient to move the mercury up to the beginning of the gold tubing. However, by closing stopcock *D* and applying heat to the glass tube by means of a tiny gas flame the expansion of the mercury can be controlled so as to force the radon into the gold capillary.

SUMMARY

Gold seeds may be made as frequently as is desired using this apparatus without disturbing the radon supply. Furthermore the accuracy with which the proper percentage of radon can be pumped into gold seeds is increased so that it is possible to make even a small number of seeds with little error.

Since the millicurie strength of the tubes to be crushed is accurately known and since the percentage of radon transferred can be precisely determined, a collection of a small number of seeds can be made with a high degree of accuracy.

Philadelphia General Hospital
Philadelphia 4, Penna.

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ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

HEAD

Hartley, J. Blair, and Burnett, C. W. F. New light on the origin of craniolacunia. *Brit. J. Radiol.*, April, 1944, *17*, 110–114.

The different theories in regard to the causation of craniolacunia are discussed and a case reported which seems to have a bearing on the subject. A child with hydrocephalus was delivered by paracentesis of the skull. There were marked lacunar changes in the anterior halves of the frontal bones at the center while the periphery and the posterior halves were normal. There was only slight abnormality in the parietal bones, which disposes of the parietal arch theory of causation. The distal portion of the squamous occipital bone derived from membrane was strikingly expanded, while the proximal segment, derived from cartilage was stunted. These findings suggest that the etiological factor was active only during the early months of ossification. It is evident that a pressure effect could not have produced this localized craniolacunia as hydrocephalus would exercise pressure equally in all directions and throughout pregnancy. An argument against the chromosomal theory of origin is furnished by the fact that craniolacunia tends to disappear with age, while chromosomal changes are never repaired. The authors conclude that this case of localized craniolacunia was caused by some dietetic defect during the earlier months of pregnancy that was remedied during the later months. Bone formed from membrane is particularly apt to be affected by such dietetic defects while bone from cartilage remains healthy. Corroborative evidence furnished by a case of fetus papyraceous is cited by the authors.—*Audrey G. Morgan.*

Hartley, J. Blair, and Burnett, C. W. F Enquiry into the causation and characteristics of cephalhaematomata. *Brit. J. Radiol.*, Feb., 1944, *17*, 33–41.

Very little attention has been paid in the roentgen literature to the diagnosis of cephalhematoma, though it is of considerable importance and these cases must be differentiated from craniolacunia and craniofenestria. The authors described 5 cases and give roentgenograms of them with roentgenograms of cases of craniolacunia and craniofenestria for purposes of comparison.

One factor in the causation of these cephalhematomas is birth pressure but such pressure acting on normal bones would not cause them, or they would be much more frequent. It is probable that the bone underlying the tumor is incompletely ossified. This theory is borne out by the fact that the majority of these cases occur in the children of primiparas in whom the head engages a month before birth. The tumor probably does not result from a single hemorrhage but from a series of small repeated hemorrhages. It is also probable that the fall in the prothrombin level in newborn infants is a factor in their causation. To detect their presence careful roentgen studies should be made before, during and after labor; examination of the blood of mothers and infants and detailed records of the appearance and behavior of infants at birth are also important. In treatment the author recommends the giving of a single dose of 10 mg. of vitamin K analogue (2-methyl-1:4 naphthoquinone) as soon as the lesion is discovered, as this dose prevents any appreciable fall in prothrombin concentration. —*Audrey G. Morgan.*

TINNEY, WILLIAM S., and MOERSCH, HERMAN J. Intracranial metastasis of carcinoma of the lung. *Arch. Otolaryng.*, March, 1944, *39*, 243–244.

Because of the lack of specificity of pulmonary symptoms and occasional absence of any symptoms referable to the thorax, the diagnosis of carcinoma of the bronchus not infrequently is extremely difficult. In some instances the problem is further complicated by early metastasis to the central nervous system, as exemplified by involvement of the nervous system in 52, or 12 per cent, of the 448 cases of proved carcinoma of the lung. The most interesting cases were the 19, or 4 per cent, in which the initial symptoms were referable to the nervous system. In 9 of these cases a pulmonary lesion was not suspected until a routine roentgenogram of the thorax was obtained. For this reason the authors wish to stress the importance of careful physical examination, including routine roentgenographic examination of the thorax, in every case in which an intracranial or intraspinal lesion is suspected.

It is often difficult and at times impossible to distinguish a metastatic lesion from a primary tumor of the brain or the spinal cord. The clinical manifestations in most of the patients suggested characteristically wide dissemination and rapid progression of the lesions and included a rather high incidence of personality changes. However, these clinical findings alone were not sufficiently characteristic in any case to be of much assistance in the differential diagnosis. The authors also wish to emphasize that in their series of cases the type of carcinoma of the lung was not a factor in the incidence of metastasis to the nervous system.—*Mary Frances Vastine.*

BROMLEY, J. F., WRIGHT, W. D., and SPIEGLER, G. The eye and radiology; symposium. *Brit. J. Radiol.*, March, 1944, *17*, 65–69.

Bromley discusses the physiology of the eye, comparing it to a photographic camera. The physicists use the foot candle as the unit of intensity at any given point and an alternative to this is the lumen: 1 foot candle = 1 lumen per square foot. The unit of brightness is the lambert which is the average brightness of a surface which emits or reflects 1 lumen per square centimeter. The brightness of emission of a fluorescent screen lies between the limits of 0.01 mililambert for thin persons and 0.001 millambert for thick patients or small fluoroscopic areas and therefore the amount of illumination of the screen is quite low and comes within pure rod function. The rods are distributed peripherally around the fovea centralis, 10 to 15 degrees away from it. Therefore to see clearly in a dim light the observer must look not directly at the object but 10 to 15 degrees away from it. The rods become tired very quickly so the observer should not look steadily at the object but allow his eyes to pass from one side of the object to the other, resting only for a second or two on points of special interest. This method of looking to one side of the object and using the "scanning" technique is important in training the night vision of pilots. Two other factors that affect visual acuity at night are good general condition and a sufficient oxygen supply. A sufficient amount of vitamin A is also necessary as lack of it causes deficiency of visual purple. Pilocarpine hastens the regeneration of visual purple after it has been bleached by bright light. Nicotine has a bad effect on rod vision. Alcohol does not affect the rods directly but depresses the higher interpretative centers.

Wright discusses the limitations of the eye and their effect on the interpretation of the fluoroscopic image. It is possible that brighter pictures may be obtained by the application of television principles to fluoroscopy. This would involve greater complexity and expense than the present method but the gain in brightness would probably justify it.

Spiegler discusses the roentgenogram from the physical viewpoint as a pattern of contrasts and shows by illustrations how the mind may lead to misinterpretation on the part of the eye. An improved system of interpretation will have to take into account the influence of stray radiation on contrast and definition to a greater degree than has been done in the past. An illustration is given showing the difference in opacity of the image of two pieces of lead, one placed on the front, the other on the back of the patient. The greater opacity of the image on the back is due to the fact that more scattered radiation reached this piece of lead.

The eye responds to different types of blurring (from movement, size of focal spot and graininess of screen) in different ways. The analysis of pictures of unknown non-medical objects is recommended as a useful way of training the mind in the reading of roentgenograms without being influenced by anticipations or prejudices.—*Audrey G. Morgan.*

McLaren, John W. Bone degeneration of the temporo-mandibular joint. *Brit. J. Radiol.*, March, 1944, *17*, 94–95.

Three types of bone degeneration may be demonstrated by roentgen examination of the temporomandibular joint: (1) erosion of the anterior and upper aspect of the affected condyle; (2) narrowing of the joint space with blurring of the bone outlines, indicating arthritic changes; (3) greater forward movement of the eminence of the condyle on the involved side than on the normal side.

Roentgenograms are given of a case in a man of thirty-nine who had complained of pain over the left mandibular joint and slight limitation of movement. Roentgen examination showed a circular area of degeneration on the superior aspect of the condyle of the jaw. Operation showed an area of degeneration just beneath the joint surface; the space was filled with a mucoid fluid. After the removal of this part of the condyle the patient had no further symptoms.

The condition is probably caused by trauma. This patient had been a sparring partner for fifteen years and had had repeated blows on the chin, but no serious injury at any time.—*Audrey G. Morgan.*

Windeyer, B. W. Malignant tumours of the upper jaw. *Brit. J. Radiol.*, Jan., 1944, *17*, 18–24.

Malignant tumors of the upper jaw are usually not diagnosed until late after the wall of the antrum has broken down. Valuable time is often lost in the extraction of teeth or treatment for sinusitis. The methods of diagnosis are inspection and palpation to detect swelling, softening or ulceration. Rhinoscopy may show a tumor bulging into the nose but may show only simple polypi. If polypi are small and multiple and bleed easily and if they occur in a patient past forty they suggest malignancy. Epitheliomata of the palate are often really tumors of the antrum. Transillumination may show opacity at the site of the disease. Roentgenography is of the greatest value in showing dullness of the antrum, the site and extent of destruction of the wall of the antrum and often also the outline of the tumor. It shows the extent of involvement of the ethmoids and other sinuses and extension to the base of the skull if it has occurred. Biopsy is of value in determining prognosis and methods of treatment. It must be taken from the deeper tissues and exploratory operation is generally necessary. To avoid increased risk of metastasis from trauma it should be performed with the diathermy electrode and the area thoroughly coagulated.

A combination of surgery and radiotherapy has proved more valuable in the treatment of these tumors than either alone. The author discusses a series of 153 cases treated by the combined method and gives roentgenograms of cases and diagrams of the method of applying radium to the antrum. Distribution of dosage is not easy to calculate because of the peculiarities in the shape of the antrum. In intracavitary radium treatment each case must be considered individually. External irradiation is first given to the whole of the affected region after the extent of the tumor has been determined. Both the 4 gm. teleradium unit and roentgen rays with a half-value layer of 2 mm. copper have been used for this purpose. Homogeneous distribution of the radiation is obtained more easily with roentgen rays and the daily sessions are shorter and therefore less troublesome for the patient. A tumor dose of from 4,500 to 5,500 r is given over a period of four to six weeks, and drainage of the antrum provided for if this has not already been done by exploratory operation. When the reaction from this treatment has subsided operation is performed. Later steps depend on the condition found at operation.

Among the 153 cases 120 were treated; the other 23, or 15 per cent, were considered too advanced or the patients refused treatment. The five year results are known only in the cases treated before 1937. Up to 1935, 69 cases were treated and 18 were symptom-free at the end of five years. In 1936 and 1937 13 cases were treated, 5 remaining free of symptons for three years and 4 for five years. From 1936 to 1939, 30 cases were treated and of these 11 were free of symptoms after three years. These figures show a marked improvement, although all the cases seen during this period were treated. Of the 9 cases of sarcoma treated before 1937 only 1 remained free of symptoms for five years.—*Audrey G. Morgan.*

Thompson, Charles M., and Grimes, Elmer L. Carcinoma of the nasopharynx. *Am. J. M. Sc.*, March, 1944, *207*, 342–348.

This paper was written to call attention to the clinical syndrome that characterizes carcinoma of the nasopharynx. It is believed that

a lack of knowledge of this symptom complex has undoubtedly been a factor in the poor control of this highly malignant disease. Every review in the literature presents an equally discouraging percentage of cures. Many authors stress the element of late diagnosis as one of the reasons for this low percentage. The following features are associated with this disease:

(1) Cervical adenopathy. Various series taken from the literature show that this occurs in from 27.5 to 77 per cent of he cases.

(2) Nasorespiratory symptoms, as evidenced by nasal obstruction, discharge or epistaxis is reported in 30 per cent of the cases.

(3) Neurological symptoms. Paralysis of one or more cranial nerves, the most frequently involved cranial nerves are V, VI, IX, resulting in facial pain, diplopia and dysphagia, respectively. These findings plus headaches are explained by direct extension of the tumor into the middle fossa of the skull. Roentgenograms of the base of the skull show a typical melted-out appearance of the sphenoid and the petrous portion of the temporal bone. Due to involvement of the cervical sympathetic chain, a Horner's syndrome may develop.

(4) Ear symptoms. As most of the tumors arise in or near the fossa of Rosenmüller, a number of auricular manifestations may be expected as early complaints. Tinnitus and a sensation of stuffiness are the other leading complaints.

(5) Duration of symptoms. It was claimed by Furstenburg that the average duration of symptoms before diagnosis was fifteen months.

Once the diagnosis is proved, irradiation to the limit of the skin and physical tolerance is the only accepted method of treatment. The general opinion is that surgery is not indicated in the treatment of these tumors.

Schmincke pointed out that these tumors arose in a lympho-epithelial region where normally considerable lymphoid tissue abounds and that they were simply anaplastic carcinomas arising from the nasopharyngeal mucosa with many lymphocytes mixed in coincidentally. They are not malignant lymphomata.

It is pointed out that profoundly ill patients can be dramatically relieved temporarily by irradiation. In several of the authors' cases, the patients were rendered symptom free for weeks or months with complete clinical regression of the primary and also the cervical node enlargement. Subsequent recurrence of the tumor or metastases has been found to be radioresistant. Therefore, once the diagnosis has been established, all patients should be treated to the limit of skin and physical tolerance even though clinically one can no longer find disease.—*James J. McCort.*

NECK AND CHEST

PAYNE, REGINALD T., TOD, MARGARET C., and LEDERMAN, M. Diagnosis, classification and treatment of tumours of the salivary glands; symposium. *Brit. J. Radiol.*, Jan., 1944, *17*, 3–12.

In any large series of tumors those of the salivary glands constitute only 1 to 2 per cent. They are of great interest, however, as it is very difficult to determine whether they are benign or malignant. Deep roentgen treatment is now used as an aid in differential diagnosis as well as in treatment. A classification of the benign and malignant forms is given and congenital hypertrophies are also included although they are not tumors, strictly speaking. Recurrence may take place many years after operation and a patient can hardly be considered free from the danger of recurrence as long as he lives.

A combination of irradiation and surgery has been found more effective than either alone. A high percentage of five year recoveries can be brought about in this way but, as mentioned above, a five year recovery is not so decisive as in tumors in other locations. Lymphosarcomas are quite radiosensitive. The mixed tumors of the parotid which are the most frequent form are less so. The best method of treatment for these is surgery followed by implantation of radium needles. The volume of tissue irradiated plays a part in the results, and the smaller the volume the greater the tolerance. That is why the tumor should be removed surgically before irradiation, which should be given in a single course within a month of operation. The incidence of recurrence also seems to be reduced when surgery is combined with irradiation. The technique and dosage of irradiation, which must be adapted to the individual case, are discussed. When surgery is impossible or contraindicated radiotherapy with radium or roentgen rays gives good results in some cases.—*Audrey G. Morgan.*

JOHNSTONE, A. S. Salivary calculus or tartar. *Brit. J. Radiol.*, Jan., 1944, *17*, 31–32.

Some confusion has arisen from the different meaning attached to the word salivary calculus by physicians and dentists. A salivary calculus

is really a concretion in one of the salivary glands or its ducts while the name tartar should be applied to surface deposits on the teeth. These may reach large size and resemble tumors. A case is described in a woman of fifty-eight who showed the changes of hypertrophic osteoarthritis. She also had a swelling of the right cheek which had been increasing in size for twenty years. An intra-oral occlusal roentgenogram showed a bone tumor apparently originating from the alveolar margin of the right maxilla. Only three teeth, the fourth, fifth and sixth, remained in the right maxilla and these were surrounded by the dense radiopaque deposit. It was removed with the three teeth which had become loosened by absorption of the alveolus. There was no evidence of invasion. The growth was diagnosed as a "salivary calculus" and removed, but without any improvement in the osteoarthritis. It was really an enormous deposit of tartar.—*Audrey G. Morgan.*

Beršack, S. R. Carcinoma of the esophagus in association with achalasia of the cardia. *Radiology*, March, 1944, *42*, 220–223.

Achalasia means a failure of the sphincter of the esophagus at the cardia to relax. It results in an accumulation of saliva and food over long periods of time that is irritating and theoretically might be supposed to bring about the development of carcinoma. Rake in fact reported an incidence of 20 per cent carcinoma in 15 cases of achalasia and Gottstein an incidence of 10 per cent in 33 cases.

But among 227 patients with carcinoma of the esophagus admitted to the Edward Hines Hospital, Hines, Ill., the carcinoma was associated with achalasia in only 1 case. The history of this case is given. This low incidence of carcinoma associated with achalasia is all the more noteworthy because shallow ulcerations, nests of epithelial hyperplasia and wart-like formations have been seen repeatedly in achalasia. Frequently small irregularities and filling defects are seen in the dilated esophagus but they may be caused by retained secretion and food particles. Roentgen examination of the esophagus should not be made until after the retained contents of the esophagus have been evacuated.

In order to show definitely any relationship between achalasia and carcinoma of the esophagus it would be necessary to prove statistically that carcinoma is decidedly more frequent in cases of achalasia than in the general population of the same age group. No such statistics are available—in fact, there are no comprehensive statistics for achalasia. This group of cases would seem to argue against any connection between the two conditions.—*Audrey G. Morgan.*

Schwartz, Leo. Adenoma of the trachea and the bronchus. *Arch. Otolaryng.*, March, 1944, *39*, 231–242.

Statistics disclose that there is actually a predilection of primary tumors for the lungs and bronchi and that 12 to 15 per cent of all cancers originate in them. In the light of recent knowledge it is found that 5 to 10 per cent of all primary endobronchial tumors are benign.

Histological Origin of Primary Endobronchial Tumors. The adenomas probably arise from the gland cells or from the cells of the gland ducts. Originating as they do in the submucosa, they lift the mucosa over their surface as they grow; therefore, the mucosa presents itself to the endoscopist's eye as the smooth glistening surface of the tumor. As the tumor grows and the lumen of the trachea or bronchus becomes partially or totally filled with the mass, molding and encasing pressure is exerted on it by the rigid bronchial or tracheal cartilaginous frame, imparting to the tumor a rounded and elongated form.

The author wonders whether this growth in the female might not be distinct from that occurring in the male and, in fact, arise from primordial gonadal cell rests. The recurring hemoptyses that are frequently concurrent with the menses, as well as the fact that these patients frequently give a history of pulmonary bouts beginning at the menarchal period, seem to support this conjecture.

Tumor Growth and Extension. Three types are described: (1) pedunculated type which, as a rule, grows entirely within the lumen; (2) intramural type which presents a flat base and grows between the cartilaginous rings; (3) endoextrabronchial or dumbell type in which a constriction takes place at the bronchial wall. The smaller portions present endobronchially and the large, extrabronchially.

Symptoms and Physical Signs. Adenoma presents no findings which are peculiar to itself. The findings are those characteristic of bronchial obstruction and depend on the location

of the tumor, the type of obstruction produced, the amount of tributary alveolar tissue obstructed and other factors, such as the type and the virulence of secondary infection.

Roentgenologic Manifestations of Bronchial Obstructions. These depend on the type of obstruction produced: (1) Stop valve obstruction—the bronchus is corked and the roentgenologic findings are those of atelectasis. (2) By-pass valve obstruction—air can pass in and out of the lung but only in limited amounts. The asthmatoid wheeze can be heard at the open mouth. (3) Check valve obstruction—the occluding body interferes with either the inspiratory or the expiratory phase of respiration leaving the other phase unimpaired. The roentgenologic findings can therefore be either those of atelectasis or emphysema.

Differential Diagnosis. The lesion with which bronchial adenoma is most frequently confused is bronchogenic carcinoma. Adenoma is overwhelmingly a disease of young women. Carcinoma is preponderantly a disease of old men. Most cases of adenoma in women occur during the years of fertility. There is some evidence that adenoma at times undergoes cancerous transition. Hemoptyses occurring with adenoma are inclined to be much more profuse than those with carcinoma and are frequently encountered during the menstrual period.

Treatment. The tumors that arise from stalk-like bases and have neither mural nor extrabronchial extensions can be entirely cured by bronchoscopic means. Those tumors which show mural and extramural extension and all cases complicated with irreparable pulmonary damage as a result of prolonged obstruction demand surgical intervention.—*Mary Frances Vastine.*

SHINALL, HAROLD L. Roentgen diagnosis of bronchiogenic carcinoma. *Radiology*, March, 1944, *42*, 213–219.

About 15,000 persons die in the United States every year of bronchiogenic carcinoma. The diagnosis is very difficult, neither clinical nor roentgen signs alone being sufficiently definite to establish it. But with careful clinical examination and absolutely accurate roentgen technique diagnosis can be made in a maximum of 87 per cent of the cases from a combination of these findings.

The authors discuss 40 cases seen at the St. Louis City Hospital in the past five years in which both roentgen and clinical examination was made. Among these the clinical examination was sufficient for diagnosis in only 6 cases, or 15 per cent, while diagnosis was made from both clinical and roentgen signs in 16, or 45 per cent.

There are three types of bronchiogenic carcinoma—those arising from the lining epithelium, those arising from the mucous glands of the submucosa and those arising from the pulmonary alveoli. Only 1 case suggesting the possibility of an alveolar origin was seen. Roentgenograms of the different types of cases in different areas of the lung are given.

The clinical features which aid in diagnosis are the age of the patient which is usually over forty years, the average in these cases being fifty eight years; the duration of the illness, three months or more; the cough usually dry and non-productive or there may be none at all; expectoration of blood at some time during the illness; loss of weight usually out of proportion to the lung involvement. There is no fever or other signs of infection unless secondary infection takes place. The chief roentgen sign is a unilateral, rounded nodular shadow, usually in the hilar region.

Fariñas holds that when a chest roentgenogram shows an expiratory emphysema, an atelectatic zone in any field, or a dense shadow in the hilar region serial bronchography is indicated for the detection of bronchiogenic carcinoma.—*Audrey G. Morgan.*

WEINSTEIN, MANDEL, and TYAU, STEVEN. Intrabronchial spread following thoracoplasty. *Am. Rev. Tuberc.*, March, 1944, *49*, 238–250.

Since the advent of surgical collapse of pulmonary tuberculosis, postoperative tuberculous spread has been a serious complication. Aspiration of infectious bronchial secretions during and following operation is the prime factor concerned. In the hours immediately following operation, cough, expectoration, and respiratory, ciliary, and peristaltic movements of the bronchi are abolished or diminished. This encourages bronchial stasis, plugging of bronchopulmonary segments, and atelectasis. As might be expected, the patients with more extensive disease and more cavitary exudate are more prone to postoperative spread.

Nineteen postoperative spreads in 14 patients are reported by the authors. This tuberculous extension represents an operative incidence of approximately 10 per cent—198

thoracoplasties having been performed in the period represented by this report. Negroes develop spread twice as frequently as whites; moreover, 4 deaths, all Negroes, resulted from the spreads—a mortality of 2 per cent. Tuberculous spreads involve twice as many contralateral as homolateral lungs while bilateral extension occurred only in 4 cases. All patients with mild spreads recovered; as might be anticipated the severe spreads accounted for all postoperative fatalities.

No single or multiple procedures can always prevent tuberculous spreads, but maintaining the tracheobronchial tree free from much secretion will help by preserving open airways and precluding the movement of infected tuberculous exudate throughout the respiratory system.—*John R. Hannan.*

Hamilton, Joseph G., Soley, Mayo H., Reilly, William A., and Eichorn, Karl B. Radioactive iodine studies in childhood hypothyroidism. *Am. J. Dis. Child.*, Nov., 1943, *66*, 495–502.

The authors employed labeled (radioactive) iodine to determine the ability of the thyroids in two groups of hypothyroid children to concentrate iodine.

They summarize their study as follows:

"The thyroids of children with hypothyroidism and without goiters are able to concentrate only small amounts of orally administered iodine, as compared with the thyroids of normal children or adults. In these hypothyroid children, the remaining thyroid tissue concentrated a larger percentage of a dose of 0.1 microgram than of a dose of 14 mg. of iodine.

"The thyroids of two children with goiters and with severe hypothyroidism took up relatively large amounts of iodine and by chemical analysis contained both thyroxin and diiodotyrosine in considerable quantities. The uptake curves of the thyroids as measured in situ were similar to those seen in hyperthyroidism in adults. Radioautographs made from sections of the thyroids of both of these children indicate that by the fifth day after the oral administration more of the iodine was in the cells than in the colloid."—*R. S. Bromer.*

Gellis, Sydney S., and Peters, Michael. Mumps with pre-sternal edema. *Bull. Johns Hopkins Hosp.*, Oct., 1944, *75*, 241–250.

The authors report an incidence of presternal edema complicating epidemic parotitis in 6 per cent of 502 patients at the Station Hospital, Fort Benning, Georgia. The condition appears to be of no particular consequence since only 5 patients developed symptoms coincident with the edema—2 had dysphagia for twenty-four hours, 1 had hoarseness and 2 were tender to palpation over the sternum. All patients having this complication were afebrile at the time of onset except those who already had orchitis. The incidence of orchitis in the 30 cases of edema was no greater than in the remaining 472 cases. Roentgen studies showed no demonstrable intrathoracic changes, though the thickening of the presternal tissues was seen in the lateral roentgenograms. The edema is central, the shape of a necklace, obliterates the sternal notch and is variable in extent. In their cases it developed between the third and twelfth days of illness and lasted from two to eighteen days. They consider it due to obstruction of lymphatics by swollen salivary glands.—*Angus K. Wilson.*

Hurst, Allan, Bassin, Sydney, and Levine, Ida. Miliary densities associated with mitral stenosis. *Am. Rev. Tuberc.*, March, 1944, *49*, 276–285.

Miliary densities associated with rheumatic mitral stenosis have received little attention in the medical literature. Recent textbooks of roentgenology state that this finding is encountered occasionally in cardiac congestion, particularly secondary to mitral stenosis, but failed to stress the not infrequent permanency of the pulmonary changes.

The authors present 5 cases of bilateral, symmetrical, diffuse, miliary, and submiliary densities associated with mitral stenosis—cases selected as representative of most of the pathological changes occurring in this condition. The 5 young men were practically asymptomatic and without auscultatory pulmonary signs when a chance roentgenogram disclosed the abnormal densities. These densities are ascribed to passive pulmonary congestion with hypertension of the lesser circuit. The vessels dilate, become engorged, and finally hypertrophy. When seen on end, these vessels are revealed as rounded densities and on horizontal section are linear. In addition, some larger, denser nodules are seen at the periphery of the lower lung fields in several cases. These are attributed to perivascular fibrosis with deposition of hemosiderin

pigment from extravasation of blood. Occasionally, numerous calcific densities (thought to be due to breakdown and subsequent healing of multiple, small, stasis infarcts attendant upon severe passive congestion) are scattered throughout the lung fields.

In spite of the obvious cardiac condition, these densities are frequently imputed to and must be differentiated from the pulmonary lesions of sarcoid, miliary tuberculosis, pneumoconiosis and lymphangitic carcinomatosis.—*John R. Hannan.*

BAER, RIDGELY W., TAUSSIG, HELEN B., and OPPENHEIMER, ELLA H. Congenital aneurysmal dilatation of the aorta associated with arachnodactyly. *Bull. Johns Hopkins Hosp.*, June, 1943, *72*, 309–331.

The authors report 2 cases in which they found extraordinary aneurysmal dilatation of the ascending aorta in association with arachnodactyly. They recognize the report of Ellis that approximately one-third of patients having arachnodactyly also have congenital malformations of the heart, and refer to their own previous report of interauricular septal defects, but the condition found in these 2 cases was unique in their experience. In addition to several non-contributory congenital defects (ectopia lentis, deformities of spine, and, in Case 1, of the thoracic cage), they found dilatation of the ascending aorta and cardiac enlargement. Roentgen examination by films and barium suspension failed to give any indication of the aortic enlargement. The first patient died when fourteen years of age, after developing auricular fibrillation, and the second patient died suddenly at the age of twenty-six years, after angina-like attacks of precordial pain.

The autopsy findings in both patients were remarkably alike. The enlargement of the ascending aorta was found to be due to changes affecting the elastica and muscular components. In some places the elastic fibers were frayed, in others condensed and in still others were totally absent. The muscle bundles, instead of the normal parallel distributions were irregularly branched, rarefied and between the branches there were large cystic spaces filled with a blue-staining coagulum. The medium was still further distorted by numerous irregular blood channels. There were small foci of lymphocytes in the adventitia but the authors felt they were not indicative of inflammatory process. There was no cellular exudate in the medium, nor was there any evidence of necrosis or degeneration.

After discussing Erdheim's "medial necrosis idiopathica cystica," which is considered due to necrosis and senile degeneration they conclude that in their cases the aneurysmal dilatation of the ascending aorta is a congenital anomaly due to abnormal development of the elastic and muscular layers. The cystic spaces filled with mucoid material are normally present in the fetus up to about three months, thereafter gradually disappearing.

The article is well illustrated with reproductions of roentgenograms, in posteroanterior projections, in support of the observation that the dilatation of the ascending aorta was not demonstrable, by excellent photographs of gross specimens and photomicrographs.—*Angus K. Wilson.*

FREEDMAN, PAUL, LEVINE, SAMUEL, and SOLIS-COHEN, LEON. Hemothorax in blood dyscrasias. *Am. J. M. Sc.*, May, 1943, *205*, 692–696.

The authors report 2 cases of hemothorax which were admitted to the Jewish Hospital in a period of two months. Both occurred in males, one due to hemophilia and one due to thrombocytopenic purpura. Both patients were in the third decade of life. They are classified as non-traumatic hemothorax. In neither was it a primary manifestation of the disease. In the hemophiliac it was acute in clinical onset, while in the purpuric it was more insidious. The patient with hemophilia eventually recovered; the patient with purpura succumbed to infection in the form of an empyema. It was thought that this infection was secondary to thoracenteses and the danger of this complication is emphasized.

The treatment of the non-traumatic hemothorax is first to care for any shock that may be present. As the bleeding undoubtedly is diffuse and not focal in origin, there is probably never an indication for thoracotomy and vessel ligation. Aspiration is usually indicated after about forty-eight hours as blood left in the pleural cavity may form fibrin clots, the latter requiring thoracotomy The entire collection should be removed in one or two aspirations as the fewer the punctures the less danger there is of infection.—*James J. McCort.*

Bannen, J. E. Post-operative pneumoperitoneum. *Brit. J. Radiol.*, April, 1944, *17*, 119–121.

There is more or less postoperative pneumoperitoneum after every abdominal operation. One causative factor is probably the habit some surgeons have of pulling up the abdominal wall to the last stitch which increases the air content of the abdomen. The conditions could be prevented or minimized by exerting gentle pressure on the abdominal wall before inserting the last stitch and in this way expelling the residual air. The free air is generally absorbed in seven days and is therefore harmless, but it may cause a more or less serious degree of respiratory embarrassment and may be mistaken for more serious conditions such as subdiaphragmatic abscess and perforated gastric or duodenal ulcer. A correct differentiation from these conditions may prevent a serious operation. Roentgenograms should be taken within forty-eight hours after an operation with the patient in the Fowler position. In this position the free air in the abdomen rises to the subdiaphragmatic region and can be seen as an aerated space 1 or 2 inches thick between the diaphragm and the liver and stomach. Roentgenograms of a case are shown.

Marked degrees of pneumoperitoneum are more apt to occur in gynecological operations where the patient has been changed from the Trendelenburg to the Fowler position and where a large amount of tissue has been removed as in hysterectomy. Postoperative pneumoperitoneum is always bilateral and unless lung complications have occurred the lung fields are clear and there is no irregularity of the diaphragm. When the condition is caused by gas-forming organisms, as in peritonitis and after a perforation, the diagnosis must be based on the acute history and the presence of gas in the abdomen before operation. The postoperative abdominal discomfort frequently attributed to gas distention of the bowel may be due to pneumoperitoneum and in that case enemas will do no good.—*Audrey G. Morgan.*

Molony, Clement J. Postoperative pulmonary collapse in childhood. *Am. J. Dis. Child.*, Sept., 1943, *66*, 280–301.

Molony presents 21 cases of postoperative atelectasis in children. The cases were collected from the records of two large children's hospitals over a period of ten years. He was not able to find in the literature an inclusive report of this condition from a children's hospital. The etiologic factors for collapse following surgical procedures are discussed and a plan of treatment based on these several factors is suggested. He emphasizes the importance of atelectasis in the production of bronchiectasis, and believes bronchoscopic drainage in cases of persistent collapse is the proper treatment.

The 21 cases are analyzed from the standpoint of incidence according to sex and age, type of operation performed, anesthetic used, symptoms presented, day of occurrence following operation, and day of clearing. In regard to the causes he discusses bronchial obstruction by a plug of mucus; the effect of hypoventilation brought about by the great decrease in vital capacity following operation; decrease in body tonus caused by the decrease in tone of the thoracic muscles and the diaphragm following operation; the role played by allergy; and the effect which may be produced by the use of the closed system of anesthesia.

He tabulates the precautions against and treatment for postoperative collapse based on the various etiologic factors. The article is profusely illustrated with roentgenograms of the changes in the lungs of many of the cases — *R. S. Bromer.*

Imber, Irving. A tumor occurring in the superior pulmonary sulcus. *Am. J. M. Sc.*, May, 1944, 207, 654–660.

This case report and postmortem study was made because it illustrates a superior pulmonary sulcus tumor in which the extrapulmonary origin of the tumor can be established, thus favoring the original contention of Pancoast that a primary tumor may arise in the pulmonary sulcus and that it may produce a definite clinical syndrome. The clinical syndrome described by Pancoast included (1) pain in the shoulder region, often radiating down the arm and eventually to the fingers; (2) Horner's syndrome; (3) local destruction of the first two or three ribs, and (4) atrophy of the muscles of the hand.

The syndrome described by Pancoast was present in this case. The clinical course from the onset of pain to death was extremely rapid, totaling only three and one-half months. Physical findings and symptoms of a pulmonary lesion, which usually have been strikingly absent in the report of cases, were present in

this case, apparently due to the large size of the tumor. The rapid growth exhibited by this lesion on roentgen examination is also somewhat unusual. None of the roentgen examinations demonstrated evidence of osseous erosion.

It was thought that the tumor mass in the pulmonary sulcus at postmortem examination was undoubtedly extrapulmonary and was so proved by microscopic section. Dr. McFarland reviewed the histologic sections of this patient. He expressed the opinion that this tumor was composed of cells which are squamous epithelial cells, but did not reach the prickle stage and that the adrenal medullary tumors and the sulcus tumors were of the same cell type. From the history obtained it is not possible to state whether the Horner's syndrome preceded the onset of scapular pain or whether it followed the onset of pain.—*James J. McCort.*

OLCOTT, CHARLES T., and DOOLEY, SAMUEL W. Agenesis of the lung in an infant. *Am. J. Dis. Child.*, May, 1943, *65*, 776–780.

The authors report a case of complete absence of the right lung in an infant two months old. The diagnosis was made during life by physical, roentgenographic and bronchoscopic examination. The roentgenographic examination showed the cardiac silhouette and mediastinal contents in the right side of the chest continuous with the shadow of the liver. The left lung showed evidence of hyperventilation. No iodized poppyseed oil entered the left main bronchus.

Deficiency of a lung has been classified as of three types by Schneider. In type 1, there is no trace of a bronchus—true aplasia of the lung and bronchus. In type 2, the bronchus is represented by a blind pouch or a nodule of cartilage and fibrous tissue and there is no pulmonary tissue—aplasia of the lung. In type 3, the main bronchus is normal in size and shape and ends in a fleshy structure—extreme hypoplasia of the lung. The third type may be impossible to differentiate from secondary retraction of a previously expanded lung. In the case reported by Olcott and Dooley, at necropsy there was no recognizable right pleural cavity. The trachea formed an almost direct line with the left main bronchus. At the site of the normal branching of the right main bronchus was a barely recognizable pocket, not over 4 mm. in any dimension and no tissue of any sort was attached to it.

Agenesis of the lung is compatible with life. It is of diagnostic importance that the chest in patients with true aplasia of the lung is usually symmetric. Pneumonia of the remaining lung is often fatal. Cases have also been reported of the remaining bronchus being occluded by a foreign body.—*R. S. Bromer.*

CHILDRESS, WILLIAM G. Tuberculosis among hospital personnel. *Am. Rev. Tuberc.*, June, 1944, *49*, 501–509.

Heimbeck reported positive skin reactions in only 52 per cent of incoming student nurses in Oslo, whereas at the time of graduation, 100 per cent of this group showed positive reactions to tuberculin.

Many other investigators have reported similar increments in sensitivity to the tubercle bacillus following contact. Workman's Compensation Acts have indicated the prevalent belief that contact with active tuberculosis provides a definite hazard—a hazard which pertains to those previously infected as well as to those not yet sensitized.

Periodic roentgen examination of the chest is considered a practical procedure, particularly for those who are required to work with tuberculous subjects; skin testing is not always practical.

This report pertains to observations conducted at the Grasslands, New York, since 1932. The bed capacity of the hospital was 800; 300 being tuberculous patients (housed in separate buildings interconnected by tunnels). The average employment numbered 750 (200 being permanently assigned to the section of tuberculosis). In all, 5,039 employees were studied, of whom 2,092 were subjected to contact with the disease.

Of the personnel without symptoms and with normal roentgen findings at the time of employment (45), 1.8 per cent of those exposed developed the disease and only 0.2 per cent of those not exposed did likewise.

Of the personnel showing active tuberculosis at the time of the first examination (14), 57 per cent had been exposed while 43 per cent had not been exposed.

Of those with a previous history of tuberculosis (65), 27 per cent of those exposed became re-activated while only 21 per cent of those not exposed did likewise.

Thus, the higher morbidities occurred among

the exposed versus the unexposed; considering all subgroupings, of 2,092 who were exposed, 3.5 per cent developed the disease, while among 2,947 without known exposure, only 0.54 per cent developed activity.

All types of personnel were concerned: physicians (13), nurses (45), attendants (10), porters (4), maids (3), kitchen workers (5), and laboratory workers (5).—*A. A. de Lorimier.*

DORMER, B. A., FRIEDLANDER, J., and WILES, F. J. Bronchography in pulmonary tuberculosis. I. Normal or questionable roentgenographic findings in lungs and positive sputum. *Am. Rev. Tuberc.*, Oct., 1944, *50*, 283–286.

The authors have analyzed 5 cases wherein sputum examinations were positive though roentgen studies were considered negative or questionable.

Anteroposterior, lateral, and oblique film studies were accomplished; thereafter, bronchography was performed. With this latter procedure, the lesion was identified in every case.

The authors state that "the early lesion in tuberculosis is nearly always a bronchiectasis or a dilated bronchus terminating in a cavity." They conjecture that in pulmonary phthisis, the basic pathology is "bronchial block with subsequent pneumonitis and either abscess or bronchiectasis."—*A. A. de Lorimier.*

DORMER, B. A., FRIEDLANDER, J., and WILES, F. J. Bronchography in pulmonary tuberculosis. II. Radiographic black-out—evaluation of underlying lesions. *Am. Rev. Tuberc.*, Oct., 1944, *50*, 287–292.

Previously, the authors have recommended bronchography for identifying the location of a tuberculous lesion when the sputum is positive though the roentgen studies be considered normal or indefinite.

This is a similar study but pertaining to cases having positive sputa and "black-out" areas, roentgenographically—opacities so great that the nature of the pathology cannot readily be discerned.

Bronchography was applied to 7 such cases where the character of the lesions could not be discerned by the conventional roentgenographic procedures. The contrast densities provided bronchographically served to visualize the character of the pathology. This procedure is recommended, therefore, as a trustworthy substitute for tomography.

Again, the incidence of bronchial occlusion and of cavity formation in tuberculosis is emphasized.—*A. A. de Lorimier.*

SHAPIRO, ROBERT. Pulmonary tuberculosis in Navy recruits. *Am Rev. Tuberc.*, June, 1944, *49*, 485–489.

It is estimated that since the last great mobilization, the Federal Government has expended well over one billion dollars toward pensioning and compensation payments of service connected disabilities due to tuberculosis.

The attributes of photofluorography are expounded with respect to its value for screening. Its application is recommended to the extent of re-examination frequently. The author suggests that when developing an acquaintanceship with this procedure, the examiner should resort to standard procedures such as the use of the 14×17 inch film, frequently in the beginning; that such a policy should provide acquaintanceship with pitfalls and artefacts and that thereafter the need for 14×17 inch films will be reduced to approximately 0.5 per cent of the cases examined.

The factor of fatigue must be considered. Each examiner should identify his own fatigue threshold, and rest accordingly. With adequate precautions, the author estimates that with the use of the 35 mm. single film, misses should be kept to within 1 to 2 per cent.

The criteria for rejection by the Navy are cited; essentially, these including: any evidence of reinfection tuberculosis; evidence of activity of the primary phase or of an extensive primary lesion such as might later produce disturbances, or evidence of fibrinous or serofibrinous pleuritis.

This analysis is based upon a study of 50,100 photofluorographic examinations of candidates for Seabee duty with the Navy (many being of the fourth and fifth decades of life—and all having passed preliminary physical examinations).

The incidence of reinfection tuberculosis was believed to be 0.87 per cent ; that of calcified primary tuberculosis, 10.7 per cent (5.3 per cent merely showing calcifications in the hilar lymph nodes; 5.4 per cent, both hilar adenopathy and calcifications in the parenchyma).—*A. A. de Lorimier.*

ABELES, HANS, and PINNER, MAX. Accidentally discovered pulmonary tuberculosis. *Am. Rev. Tuberc.*, June, 1944, *49*, 490–500.

Chest surveys accomplished by roentgenography have revealed lesions in subjects in whom no illness had been suspected. Some of these lesions may be readily identified as tuberculosis; others may be so identified with the support of corroborative studies, while some remain uncertain as to the exact nature of the pathologic condition.

This report is based upon 91 cases of unsuspected lesions wherein the roentgen evidence was identified by corroborative hospital studies. Twenty-nine of the examinations were conducted among candidates for the Civil Service; 25 were merely contacts; 14 were candidates for employment to industry; 8 were included in a school survey; 6 were applicants for hospital employment, and the rest, similar healthy appearing adults seeking examinations in connection with insurance, premarital status, etc.

Only 67.4 per cent of the cases showing definite evidence of tuberculous activity had noticed any symptoms. The most common symptoms were of fatigability or occasional hemoptosis. Even after the diagnosis was established, in many of these cases, there was no substantial disturbance of wellbeing until as long as two years. Physical examination revealed definite findings in only 35 of the 91 cases (though 28 had minimal lesions; 40 moderately advanced, and 23, far advanced lesions). Roentgen studies indicated bilateral lesions in 47 of the cases; unilateral, in the right lung, in 31; unilateral, in the left lung, in 13. Sixteen of the cases showed definite cavities—all of which revealed tubercle bacilli when studied by direct smears or concentration tests.

The sputum was routinely examined first by direct smear; thereafter if negative, by concentration studies and possibly by culture or gastric washings. Using one or another of these methods (repeated studies being necessary in many of the cases), the tubercle bacillus was isolated in 84.6 per cent of the group.

Thirty-seven of the patients developed a white blood count in excess of 10,000; 54 showed a sedimentation rate in excess of 9 mm. for the first hour (Westergren method).

Determination of activity of a lesion was based upon: (1) roentgen evidence (serial studies, with consideration as to progression or regression of the visualized lesion)—indicative in 90.7 per cent of the group; (2) sputum studies—with demonstration of the tubercle bacilli in 89.5 per cent; (3) blood counts and sedimentation rates—indicative in about 60 per cent.

Thus, the authors emphasize the particular value of roentgen studies and bacteriological examinations and the consideration of the clinical symptoms. Among the cases of inactivity, examinations of the sputum, guinea pig inoculations, white blood counts and sedimentation rates were negative.—*A. A. de Lorimier.*

MITCHELL, EARL B., and THORNTON, T. F., JR. Lower lobe bronchiectasis associated with tuberculosis. *Am. Rev. Tuberc.*, Jan. 1944, *49*, 38–47.

The mortality from lobectomy is now reduced to 5 per cent or lower in several large institutions. Increasing numbers of patients with bronchiectasis are accepting this operation which offers greater relief in most instances than methods of medical management. One must bear in mind that a similar procedure in patients with tuberculous bronchiectasis carries with it a mortality of 18 per cent. Furthermore, such distressing complications as persistent draining sinus or fistula, tuberculous spread, and tuberculous empyema occur in about one-third of the patients. In spite of this risk, there is a definite indication for surgery if the tuberculosis seems stationary in the remainder of the lung fields and the symptoms of bronchiectasis are severe.

The fact that tuberculosis has so frequently been the underlying disease in bronchiectasis of the upper lobes and has not been found in lower lobe bronchiectasis has given rise to a widely accepted and reasonably accurate rule of thumb—that bronchiectasis limited to an upper lobe is tuberculous and limited to a lower lobe is non-tuberculous. The incidence of tuberculous bronchiectasis has not been determined accurately. The 5 cases reported by the authors represent approximately 5 per cent of all the patients with bronchiectasis seen at the University of Chicago Clinics in the past ten years.

Tuberculous lower lobe bronchiectasis should be suspected in any patient presenting one or several of the following features: (1) suggestive history; (2) roentgenological evidence of apical

tuberculosis; (3) bronchoscopic finding of bronchial stenosis; (4) unilateral or unilobar bronchiectasis. However, the diagnosis can be made with certainty only if a positive sputum is obtained. Repeated, careful sputum examinations in all patients having bronchiectasis are mandatory. If the diagnosis can be made early in the course of treatment, the authors believe that judicious use of thoracoplasty or lobectomy, or a combination of these procedures, will give favorable results in a high percentage of cases.—*John R. Hannan.*

BOBROWITZ, I. D. Round densities within cavities; lung lesions simulating the pathognomic roentgen sign of echinococcus cyst. *Am. Rev. Tuberc.*, Oct., 1944, *50*, 305–312.

Two cases are reported wherein roentgenography of the chest revealed lesions showing a round uniformly dense focus surrounded by a concentric air space and membrane.

The author states that this feature has been described in connection with the echinococcus cyst, but otherwise not defined in the literature.

The lesions were visualized by conventional roentgenography—and even more clearly by tomography.

Both cases were subjected to surgery; in one, the process was identified as inspissated pus; in the other, as a blood clot.—*A. A. de Lorimier.*

ABDOMEN

CALTHROP, G. T. Cholecystography; pheniodol as a medium for the Graham-Cole test. *Brit. J. Radiol.*, Feb., 1944, *17*, 60–61.

The chemistry of the various media used for cholecystography is discussed. Recently a new compound called pheniodol has been introduced. It is β-(4-hydroxy-3:5 diiodophenyl) α phenyl propionic acid. It was described by Dohrn and Diedrich in 1940 and placed on the market in Germany under the trade name of Biliselectan. Now a pheniodol of British manufacture is available. It is given only by mouth.

The author has tested its oral use in 100 cases. He finds it is a safe, convenient, economical and satisfactory means of examining the function of the gallbladder. The density of the shadow was generally as good as that after the intravenous injection of iodophthalein and the risks attending the use of that drug were eliminated.—*Audrey G. Morgan.*

BRYAN, LLOYD, and PEDERSEN, N. S. New gallbladder contrast medium; priodax. *Radiology*, March, 1944, *42*, 224–225.

Tetraiodophenolphthalein has been the only contrast medium used in the examination of the gallbladder but it has a tendency to cause diarrhea, nausea and vomiting. A new medium called priodax has been produced. It is β-(4-hydroxy-3,5-diiodophenyl)-α-phenyl-propionic acid. It is excreted in a few days by the kidneys and does not collect in the colon as tetraiodophenolphthalein does. The minimal lethal dose is considerably higher than that of tetraiodophenolphthalein on parenteral administration and about the same on oral administration. It causes much less distress to the patient than the latter drug and is more effective as a diagnostic agent.

It has been given to 845 patients examined for disease of the gallbladder. Among these 227, or 27 per cent, were reported to have poorly functioning gallbladders. All of the gallbladders that showed impaired function roentgenographically with the new medium were found on operation to have some abnormality. One cancer of the gallbladder was found on operation.—*Audrey G. Morgan.*

PAUL, L. W., and POHLE, E. A. Oral cholecystography; a comparative study of the single- and divided-dose method with contrast media in liquid and solid form. *Radiology*, March, 1944, *42*, 226–232.

The authors have been using sodium iodophthalein as a contrast medium in cholecystography, giving the first dose of 3.5 gm. after a regular lunch and the second dose of the same size after a fat-free supper. This gave a high degree of accuracy. There were few faint or questionable shadows.

They have now made a comparative study of this drug and the new contrast medium, priodax. They made 194 examinations on 148 patients, 114 of whom were given priodax and 80 iodophthalein. Priodax was given in a dose of 3 gm. Both media were used in 46 cases. It was found that the single dose method was as effective with both drugs as the intensified technique with a larger number of doses.

A table is given showing the results with the two drugs. The shadow-producing qualities of the two were about the same. There was slightly less nausea and vomiting after priodax but practically as much diarrhea (22.8 per cent

with priodax and 28.7 per cent with iodophthalein) though the diarrhea was somewhat more severe with the latter drug.

The simplified technique now used is to give one dose of the drug after a fat-free supper, eat no more after this and report for roentgen examination at 8 o'clock the next morning. If gas obscures the gallbladder pitressin is given; a cleansing enema may be given instead but is not as effective. Careful roentgen technique is of the greatest importance. The use of the rotating anode tube and the high-speed Potter-Bucky grid with short exposure times (one to two tenths of a second) was very helpful in giving clear, sharp shadows with sufficient contrast.

Priodax is easier to give than sodium iodophthalein as it comes in tablet form.—*Audrey G. Morgan.*

HEFKE, H. W. Cholecystography with priodax; report on 600 examinations. *Radiology*, March, 1944, *42*, 233–236.

There are decided inconveniences in the use of tetraiodophenolphthalein as a contrast medium in cholecystography. It often causes diarrhea, nausea and vomiting. Vomiting may be so severe as to cause loss of contrast material and failure of visualization. A new drug called priodax has been introduced. It has a 52 per cent iodin content and the clinical dose of 3 gm. is far below the toxic level. The author has used this drug in 600 gallbladder examinations. He found that gas and the presence of the medium in the colon, causing confusing shadows, is not as frequent with priodax as with tetraiodophenolphthalein. Priodax was much better tolerated, and diarrhea, nausea and vomiting were less frequent and less severe with its use. Three out of 100 male patients complained of some burning on urination after priodax, due to the fact that it is excreted by the kidney. But urine examination in 30 patients showed no irritation of the kidneys. There was no evidence of any injury of the patients' health by the drug.

Priodax gave as good results as tetraiodophenolphthalein and in cases of non-functioning gallbladder even better. In 30 cases in which the first examination showed non-functioning gallbladder the dose was repeated. Only 1 of these patients showed a normally functioning gallbladder on re-examination while with tetraiodophenolphthalein 1 out of every 4 patients show normal function on twenty-four hour re-examination.

Surgery was carried out in 60 of these cases and operation confirmed cholecystography in all cases in which stones had been shown. Stones were also found in 1 case in which the diagnosis had not been made. In 1 case diagnosed as non-functioning gallbladder surgery showed normal function. The percentage of accuracy in the cases checked surgically was 96.7 per cent, which compares favorably with the findings after tetraiodophenolphthalein.—*Audrey G. Morgan.*

MUNK, JULIUS. X-ray appearances in amoebic hepatitis. *Brit. J. Radiol.*, Feb., 1944, *17*, 48–53.

There are very few direct roentgen signs of amebic hepatitis and therefore diagnosis must be made chiefly from indirect signs in the right half of the diaphragm and the lower field of the right lung. Diaphragmatic changes occur only in cases in which there is upward enlargement of the liver. This may be a generalized enlargement of the liver or only the subphrenic part may be involved

Ten cases are described and illustrated with roentgenograms. From a study of the latter a division is made into three groups. In the first group in which only the lower anterior part of the liver is involved or in which the abscess is intrahepatic or subhepatic without general enlargement of the liver there are no roentgen signs. In the second group there were different degrees of total elevation of the right half of the diaphragm with different degrees of restricted movement, extending to complete cessation and with paradoxical movement on functional examination. There were also various degrees of atelectasis of the lower lobe of the right lung, pleurisy, enlargement of the intercostal spaces, blurring of the diaphragm and displacement of the heart. In the third group there were different degrees of bulging of the diaphragm, and generally restricted movement or no movement of the bulge on respiration, with increased movement in other parts of the diaphragm.

The importance of functional examination of the right diaphragm by Müller's and Hitzenberger's tests is emphasized and the different appearances of atelectasis of the lower lobe of the right lung demonstrated. The value of the plate-like atelectatic foci demonstrated by Fleischner in 1936 as a roentgen sign is shown.

A roentgenogram similar to that of amebic hepatitis may be seen in other conditions such as the initial stage of pleurisy, all conditions of increased intra-abdominal pressure, a hydatid cyst or liver metastasis on the upper surface of the liver, paranephritic abscess or bronchostenosis, but there are usually differentiating clinical signs, which are discussed.—*Audrey G. Morgan.*

HILLER, GLENN I., and JOHNSON, RICHARD M. Abdominal aortic aneurysm; rupture into the jejunum preceded by occult blood in the stool. *Am. J. M. Sc.*, May, 1944, *207*, 600–606.

The patient, a white male, aged seventy-six, was admitted to the hospital on December 2, 1942. He had enjoyed good health most of his life and until five weeks prior to hospitalization, when epigastric pain and abdominal distention occurred with abrupt onset. At times the pain was cramping, knife-like and radiated across the upper abdomen. Nausea and anorexia were prominent features and on one occasion he had vomited a large quantity of bitter tasting greenish fluid material. Since the onset of symptoms he had lost 10 lb. in weight and complained of extreme weakness and dizzy spells. Physical examination did not reveal any masses or pulsations in the abdomen and admission blood count revealed 10.5 grams of hemoglobin per 100 cc.; and 4,600,000 red blood cells per cubic millimeter. Repeated stool examinations showed the guaiac test for occult blood in the stool to be positive. Roentgenologic examination of the upper gastrointestinal tract and the colon did not reveal any abnormalities.

In view of the fact that Golden has pointed out that blood in the stools without apparent cause is an indication for study of the small intestines, it might have been well if this patient had been studied by serial roentgenograms of the small bowel or by the method of the small bowel enema as advocated by Schatzki.

The patient died suddenly two weeks after admission. At autopsy an arteriosclerotic aneurysm of the abdominal aorta with rupture and perforation into the jejunum was found.—*James J. McCort.*

BRADLEY, J. EDMUND, and GREINER, D. JAMES. Diaphragmatic hernia. *Am. J. Dis. Child.*, Aug., 1943, *66*, 143–149.

A case of the rare type of congenital diaphragmatic hernia on the right side is reported. The only content of the hernial sac was a portion of the liver. The roentgenogram of the chest showed displacement of the heart to the left side with a dense shadow occupying the lower half of the right side of the chest that was later proved to be due to the upward displacement of the liver. The condition had existed since early fetal life. This was evident by the malformation or molding of the right lobe of the liver, which projected into the hernial sac, and the adherent maldeveloped, or fetal, right lung, which was closely associated with the sac. The case was typical of this type of lesion. The symptoms were almost entirely respiratory, appeared within two weeks after birth and varied in type and intensity until the patient's death, at the age of six months.—*R. S. Bromer.*

MORRISON, HOWARD J., and NEVILLE, R. L. Omphalocele with congenital obstruction; report of case with Meckel's diverticulum. *Am. J. Dis. Child.*, May, 1943, *65*, 781–784.

Omphalocele or exocelon is a true congenital umbilical hernia, containing intestine or abdominal organs. The authors report a case of omphalocele with congenital obstruction of the intestine. The intestines could not be reduced until the infant was anesthetized. The patient was operated upon when eighty minutes of age. Convalescence was uneventful, and at the age of fourteen weeks the infant weighed 12 pounds 11 ounces and appeared perfectly normal. An unusual physiologic phenomenon of the omphalocele's doubling in size when the infant began to cry was due to a valve-like obstruction, which caused air to distend the intestines within the omphalocele. Roentgen examination at the age of five months failed to reveal the presence of Meckel's diverticulum which had been found at operation. This was probably due to the fact that the diverticulum atrophied after the operation.—*R. S. Bromer.*

LEMONE, DAVID V., and COOPER, WILLIAM GRANT, JR. Total subphrenic abscess. *Radiology*, March, 1944, *42*, 283–285.

Subphrenic abscess is generally an accumulation of pus in one or two of the numerous peritoneal pockets between the diaphragm and the mesocolon. A large number of the subdivisions of this space may be involved but in that case the patient usually dies. A case is here described in a farmer fifty-eight years of age

who fifteen years ago had had an attack of abdominal pain after eating, nausea and vomiting of bile. After recovery from this attack he was well for twelve years when he had recurring attacks of increasing frequency and severity. Five weeks before admission to the hospital he had an acute attack with prostration and violent nausea and abdominal cramps. On admission roentgen examination showed a translucent cavity involving the entire upper third of the abdomen from the diaphragm to the level of the fourth lumbar vertebra and from one lateral wall of the abdomen to the other. The entire liver, stomach and spleen were included in the cavity. The movement of the stomach was rapid and the barium mixture passed promptly through an opening in the duodenum into the cavity. After gastric lavage the abscess cavity was almost entirely cleared of the barium mixture indicating a free fistulous tract at the site of the perforation.

After closure of the fistula and drainage the patient recovered. Roentgenograms and a diagrammatic sketch show the findings.—*Audrey G. Morgan.*

PASTERNACK, JOSEPH G. Calcareous pancreatitis. *Ann. Int. Med.*, Nov., 1943, *19*, 757–767.

In a discussion of the etiology the theory is presented that in acute pancreatic necrosis the fat necrosis has been shown to be due to the splitting of neutral fat into fatty acids and glycerin by the action of lipase that has escaped into the tissues from the pancreatic juice. The glycerin has been absorbed and the fatty acids combined with calcium to form insoluble soaps.

The consequences of calculi and calcareous deposits are atrophy and progressive fibrosis with more or less destruction of the parenchyma of the gland. As a result of obstruction of the ducts or destruction of the acinar tissue disturbances due to a deficiency of the pancreatic enzyme in the intestine may be conspicuous. Diabetes occurs when enough of the islands of Langherhans are destroyed. In 1 of the cases here presented a fragment of stone was found in the ampulla of Vater.

The clinical diagnosis of calcareous pancreatitis depends upon the roentgenographic demonstration of calcification in the pancreatic region. Pancreatic stones are usually sufficiently radiopaque to cast a shadow. When the clinical disease is not suspected shadows of stones in the pancreatic region may go unheeded and without proper interpretation. In the present series 2 patients died of tuberculous bronchopneumonia and 1 of pulmonary abscess and gangrene. The fact that patients with prolonged pancreatic insufficiency are predisposed to pulmonary tuberculosis, suppuration and gangrene has been previously noted.

As regards treatment when the symptoms are principally those of pancreatic achylia, pancreatic enzyme therapy is highly effective. The response is characterized by a decrease in the frequency and bulk of the stools, associated with gain in weight and increased strength.—*James J. McCort.*

KAUFMANN, WILLIAM, and CHAMBERLAIN, DOROTHY B. Congenital atresia of pancreatic duct system as a cause of meconium ileus; critical review of the literature, with a report of one case. *Am. J. Dis. Child.*, July, 1943, *66*, 55–67.

The authors report a case of meconium ileus in a three day old infant. Their patient, a white girl, born prematurely. Necropsy and further study of the pancreas, which was removed, established the absence of any intrapancreatic ducts, which made it probable that the excretory duct was also absent. The cause of the meconium ileus in their case seemed to be similar to that in the cases reported by Kornblith and Otani and others. Nevertheless, the fundamental lesion was somewhat different from that in cases hitherto reported, in that there was complete maldevelopment of all intrapancreatic ducts, ductules and acini while in all other cases of this type the ducts were present and often cystically dilated (cystic fibrosis).

The mechanism involved in the production of meconium ileus is thought to be as follows: Obstruction of the larger pancreatic duct, atresia of the pancreatic duct system or malformation of the entire exocrine system prevents the exocrine secretions of this gland from reaching the intestine. Acinar atrophy and fibrosis may result from the two first anomalies. Normal meconium, which is a dark brownish green, semisolid, viscid substance composed of mucus, bile, intestinal secretions, fat globules, cholesterol, vernix caseosa, epithelial cells and hair, requires pancreatic enzymes as well as bile for the maintenance of its normal composition and consistency. In

case the pancreatic enzymes are lacking in the intestine, a failure of digestion of fats and proteins in the meconium with resulting inspissation and hardening will occur. The tough, semisolid meconium accumulates in the intestine, since normal intestinal peristalsis cannot dislodge it, and fatal intestinal obstruction is the outcome.

Two greatly distended loops were found in the proximal part of the ileum. These loops were filled with thick, mucoid, sticky, dark green meconium which could be forced out of the lumen only by continued firm pressure. Its consistency was like putty. The enlarged portion of the ileum tapered off gradually until it averaged 1 cm. in diameter at the ileocecal valve. The latter was probed and was found to be patent.

The roentgen examination of the patient showed, after barium sulfate was introduced into the stomach through a tube, no definite dilatation of the stomach. The barium passed immediately into the duodenum. Two hours later the barium had appeared in the upper loops of the jejunum. Just distal to this, with the patient in the inverted position, a loop of small intestine was visible. There were multiple fluid levels in the gas-distended intestinal loops. A barium sulfate enema filled the rectum, the sigmoid flexure and the distal half of the descending portion of the colon. It was impossible to fill the remaining portion of the large bowel. A diagnosis of intestinal obstruction due to possible atresia of the lower part of the intestinal tract or due to compression from without was made.

The literature is reviewed in complete fashion and the possible causes of meconium ileus are discussed in detail.—*R. S. Bromer.*

Miller, Edwin M., Greengard, Joseph, Raycraft, William B., and McFadden, Irma. Congenital atresia of the duodenum and of ileum; report of two cases with successful results following operation. *Am. J Dis. Child.*, Sept., 1943, *66*, 272–279.

The authors report 2 cases of congenital atresia of the ileum in which they performed successful operations. They also discuss congenital obstructions of the duodenum and the ileum (small bowel), contrasting the clinical pictures of the two conditions and emphasizing the essential points in the treatment of both.

They state that progress in the surgical correction of congenital obstruction of the duodenum and ileum can be made only by close observation of the newborn infant during the first few days of life. Vomiting should not be considered as necessarily due to improper feeding. If it is persistent and remains constantly biliary, it is quite likely due to congenital obstruction of the duodenum. If vomiting persists and the material becomes brown and is associated with a progressive distention of the abdomen, there is in all probability a congenital obstruction of the lower portion of the ileum. In either case a roentgenogram of the abdomen may at once settle the diagnosis, and if it is followed by immediate accurate surgical measures there is a very fair chance of an excellent result.—*R. S. Bromer.*

Porter, D. C. Megacolon. *Brit. J. Radiol.*, April, 1944, *17*, 132.

True megacolon is comparatively rare. Two cases are described and illustrated with roentgenograms. The first was in a girl of seventeen with multiple congenital anomalies including slight shortening of the leg and an imperforate anus which was opened thirty-six hours after birth. The sigmoid condition was probably also congenital. She was extremely constipated until a lumbar sympathetic ganglionectomy was performed, since which time bowel action has been normal but the anatomical structure of the bowel did not return to normal.

The second patient was a woman of fifty-nine who has been constipated for as long as she can remember. The bowels act at intervals of four to five days and for several years the stools have been the thickness of a pencil. She has no pain on defecation and there are no signs of toxemia. A roentgenogram is given showing the difficulty of evacuating the dilated bowel in spite of daily colonic lavage.—*Audrey G. Morgan.*

GENITOURINARY SYSTEM

Kirsh, David, and Diaz-Rivera, R. S. Perinephric abscess—a previously unreported complication of amebiasis. *Am. J. M. Sc.*, Sept., 1943, *206*, 372–378.

After a thorough search of the American, English, and Spanish literature, the authors were unable to find a single instance of perinephric abscess complicating amebic dysentery. The urinary complications of amebiasis are said

to be cystitis, pyelitis, nephritis, urethritis, and kidney abscess.

The case reported here is that of a forty-seven year old Italian male who was admitted complaining of pain in the right side of the back and of the upper abdomen. For the past nine years he had suffered from relapses and exacerbations of a bloody diarrhea, accompanied on occasions by severe cramping abdominal pains which, in many instances, simulated appendicitis. Four months before admission the diarrhea recurred to a moderate degree with four to seven bowel movements a day, which on occasions consisted of only a small amount of mucus. On physical examination the liver apparently extended 17 to 18 cm. below the fifth rib. It, and the right costovertebral angle, were exceedingly tender. The stool examination was negative for *Endamoeba histolytica*. Roentgenographic studies revealed a slight elevation of the right diaphragm with haziness at the base of the right lung. The liver was reported not enlarged.

Eight days after admission laparotomy showed that the liver was diffusely enlarged but no pus or interhepatic or subdiaphragmatic masses were encountered. By blunt dissection carried around retroperitoneally to the perinephric space a large collection of pus was opened and drained. The cavity occupied the entire perinephric space. The pus was thick and yellow and was drained through a lateral stab wound. Ten days after operation blocked paraffin sections of the pus revealed *E. histolytica* in the vegetative form and this finding was confirmed three days later. The patient responded rapidly to a course of emetine hydrocloride. The authors feel it is possible that the parasite travelled through the lymphatics and localized in the perinephric space.—*James J. McCort.*

SHRADER, J. C., YOUNG, JOHN M., and PAGE, IRVINE H. Pyelograms in patients with essential and malignant hypertension. *Am. J. M. Sc.*, April, 1943, *205*, 505–514.

This study was made to find the answer to two questions: (1) Is there a pyelogram characteristic of hypertensives? (2) Is the incidence of abnormal urograms in an unselected group of hypertensives greater than in an unselected group of normotensive controls? This study did not include hypertensive patients who showed evidence of surgical renal disease such as stone, tumor or infection.

The following definitions are given: the "intrarenal" pelvis is one in which the renal tissue wholly surrounds it and the ureteropelvic juncture is seen in the roentgenogram as part of the medial border of the shadow of the kidney. The ureteropelvic junction in a kidney with a "hilar" pelvis is separated from the medial border with the pelvis partly within it. The "extrarenal" pelvis appears to be outside the kidney, making the interrenal portion seem to consist entirely of calyx. Since the kidneys occupy a position of varying obliquity in the abdomen and the roentgenograms were taken in the conventional anteroposterior exposure, it is difficult to see how this classification can have any scientific value since the factor of projection is entirely ignored.

Each pyelogram was inspected for the following characteristics:

1. Location of renal pelvis as to whether it was intrarenal, extrarenal, or hilar.
2. Level of renal pelvis in relation to vertebral landmarks.
3. Renal torsion; complete or incomplete rotation.
4. Presence or absence of a right-angled ureteropelvic junction.
5. Capacity of the pelvis as judged from the comparative size of the shadow.
6. Size of calyces in relation to pelvic size.
7. Number of minor calyces.
8. Configuration of calyces and infundibula.
9. General shape of pelvis; triangular, square or rounded.
10. Axis of renal pelvis; normal oblique, horizontal or vertical.
11. Tendency toward intrarenal duplication of pelvis (bifid pelvis).

The upper limits of normal arterial pressure was arbitrarily taken as 149 mm. systolic and 89 mm. of mercury diastolic. The retrograde pyelograms of 100 hypertensives and 100 normotensives were studied. The retrograde pyelograms were made by the gravity method.

It was concluded from this study that the retrograde pyelogram of patients with essential hypertension does not differ significantly from those of normotensives. The incidence of urographic abnormalities in an unselected group of hypertensives appears to be no greater than in normotensives.—*James J. McCort.*

BARDEN, STUART P. Two unusual cases of urinary tract calculi. *Radiology*, March, 1944, *42*, 285–288.

Two cases are described in which a roentgenogram of the abdomen showed multiple smooth, round shadows arranged in groups but not in the usual position for urinary tract calculi. In the first case the patient, a man of twenty-three, had gastrointestinal symptoms but no urinary symptoms. Physical findings and urine examination were negative. Intravenous urography showed absence of kidney function on the left side and a retrograde study finally showed a huge hydronephrotic sac in this region that contained multiple calculi. The patient recovered after nephrectomy. The specimen showed nephrolithiasis, hydronephrosis and a congenital polar vessel that caused constriction of the pelvis at the ureteropelvic junction. This case shows how long a urinary tract lesion may exist without causing urinary symptoms and that urinary tract disease is often manifested by gastorointestinal symptoms.

In the second case a man of twenty-eight had intermittent pain in the left loin radiating down the groin and into the left scrotum and pain in the hip, relieved by rest. Three years before he had had hematuria for a short time. There was no palpable abdominal mass and no tenderness in the loin. A survey roentgenogram of the abdomen showed multiple round grouped opacities in the midline overlying the sacrum. Intravenous urography showed a left obstructive uropathy, the point of obstruction by multiple calculi being at the distal third of the left ureter. Ureteral catheterization and retrograde pyelography showed that the calculi were in the ureter. Ureterotomy was performed and 72 stones removed. Afterward dilatation of the ureter was performed and the patient is in satisfactory condition.—*Audrey G. Morgan.*

SKELETAL SYSTEM

LEINWAND, IRVING, and DURYEE, A. WILBUR. Chronic hypertrophy of the skin and long bones: an osteo-dermopathic sydrome. *Ann. Int. Med.*, Dec., 1943, *19*, 1018–1028.

The characteristics of this sydrome are: (1) It affects young adults, beginning before the twentieth year, and is most apparent between twenty and thirty. (2) It is limited to males. (3) It is not related to occupation and there is no familial history or history of syphilis. (4) The general health is not affected. (5) The skin becomes thick and furrowed, especially over the forehead, face, scalp, hands and feet. No other parts of the body are affected. (6) There is a thickening of the periosteum which is bilateral and symmetrical. (7) The extremities show considerable hypertrophy due to thickening of the skin and periosteum. (8) The hands are enormous. (9) There is a normal mental state and no loss of libido. In the patient presented here the condition began, about nine years prior to admission, with pain in the left ankle. A roentgenogram of the lower half of the tibia and fibula showed marked periosteal irregularity of both the tibia and fibula. There was no significant change in this appearance during the period of observation. A biopsy of the tibia was taken and the sections of decalcified bone showed a porositic cortex with the marrow spaces enlarged. The periosteal surface suggested some slight previous activity, i. e., young periosteal cells but no osteoclasts, now quiescent. The histological diagnosis was chronic productive inflammation of paraperiosteal tissues, paraosteitis and osteoporosis.

The differential diagnosis included hypertrophic pulmonary osteoarthropathy, familial acromegalic skeletal disease, acromegaly, osteitis deformans, chronic venous statis, and syphilis. The presence of an excessive amount of estrogen in the male is suggested as a possible etiological factor in the syndrome of this patient.—*James J. McCort.*

RILEY, CONRAD M., and SCHWACHMAN, HARRY. Unusual osseous disease with neurologic changes; report of two cases. *Am. J. Dis. Child.*, Aug., 1943, *66*, 150–154.

The authors state the purpose of this report is to call attention to 2 unusual yet similar cases in which bizarre osseous changes and hyperreflexia were the outstanding features. They have been unable to find similar cases either in the literature or by consultation with persons versed in pathology of bone. They hope the report of the cases will call to light others and add to the understanding of the condition.

Both children, the subjects of the report, had a peculiar gait which was characterized by a straight leg, a wide base and a calcaneus limp; neither child could run. Ankle clonus and hyperreflexia were present in both children. One child showed extreme anorexia with emaciation; the other showed anorexia only. Both children fatigued easily. Roentgenograms of the long bones were similar, showing sym-

metrical areas of increased density and dilatation of the bony shafts with scattered areas of diminished density involving all the long bones. The base of the skull in one case was greatly increased in density. A biopsy of bone in the other case showed concentration of organic matrix and minimal evidence of bone resorption. There was no evidence of an inflammatory process.

In view of so many findings in common in the 2 cases, the authors regard it reasonable to suppose that in both there is the same underlying disease process.—*R. S. Bromer.*

ROBERTS, F. Nature and functions of the intervertebral discs. *Brit. J. Radiol.*, Feb., 1944, *17*, 54–59.

Schmorl's work, based on the examination of some thousands of normal and pathological spines, gave the first thorough study of the intervertebral discs. The author says there is no denying the anatomical facts that Schmorl brought out but there is room for differences of opinion in regard to the mechanical and physiological principles on which he bases his conclusions. He discusses these principles critically and states his own conclusions when they differ from those of Schmorl.

He says there is no foundation whatever for the belief that the nucleus pulposus has any power of expansion. Nor is it elastic in the true sense of the word, which means that a body has the power to recover its original shape when a deforming force is withdrawn. The nucleus does protrude when it is bored into but this is due to tension of the capsule. It also bulges when the joint is opened horizontally, but this is due to tension of the fibers which make up its mesh. In these cases both internal and external forces are at work and it is not a true elasticity.

The presence of the nucleus makes it possible for pressure to be transmitted evenly from the lower surface of one vertebra to the upper surface of the next. The discs do not act as cushions when subjected to a vertical force unless they are flattened by that force, and this is highly improbable. But they do act as cushions in the sense that the fluidity of the nucleus permits of a passive change in the shape of the discs and therefore in the shape of the spine as a whole. This enables the muscles to take the strain of any sudden movement. In addition the discs act as distributors of tension. The tonus of the capsule keeps the nucleus under tension.

The evolution of the spine is discussed and, unlike Schmorl, the author believes there is no evidence that the assumption of the erect position has placed any additional strain on the spine.

Apparently these facts have little to do with radiology but the author believes the radiologist should study the function as well as the form of the structures he examines.—*Audrey G. Morgan.*